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RELATION OF NITROGEN BODIES OF BLOOD TO SURGICAL PROBLEMS IN LIVER AND IN BILIARY TRACT DISEASE

I. INTERRELATIONSHIPS OF NITROGEN BODY METABOLISM AND CRITERIA ON WHICH JUDGMENTS ARE TO BE BASED *

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Retention of nitrogen bodies in the blood stream is the most important characteristic of a form of general intoxication of the body which is a common factor in a number of clinical conditions associated with disease of the liver, with disease of the intestinal tract and with disease of the urinary tract. Obstruction to the natural outflow of the normal excretions in each of these three sets of organs is frequently found, clinically, with this form of general intoxication. Evidently there is a common cause for the development of this intoxication, or a group of causes which, because of a common mechanism in the normal interrelated functions of these generally allied groups of organs, results in a common secondary lesion to which the retention of nitrogen bodies in the blood is due.

A cursory view of the facts available in this general class of disease brings the conviction that this is an extremely complicated problem or group of interrelated problems, and it is apparent that a satisfactory elucidation of the mechanism present and of the causes for this general form of intoxication can follow only after the normal mechanisms and processes are completely understood. This is the purpose of the preliminary article.

ANATOMIC PATHS

The chart is a graphic representation of the general interrelationships of three allied groups of organs: the liver, the intestinal tract and the systemic circulation.

The first stage in the absorption of the products of digestion of nitrogenous foodstuffs from the alimentary canal takes place from the small intestine into the portal circulation and into the thoracic duct. The lymphatic flow in the latter path communicates directly with the systemic circulation.

* From the Mount Sinai Hospital.

That portion of the products of digestion which pass into the portal circulation primarily pass through the liver. In the liver, part of the material is changed in, or by the agency of, the liver cell, and the products of this change pass out in the biliary tract in the bile while the remainder passes into the systemic circulation.

The liver represents a powerful agency for discrimination between the preceding part of the picture (that is, the portal area) which represents the height of production and the succeeding part of the picture which represents elimination. In the liver, a separation occurs between those bodies obtainable from the food intake which are useful to the body economy and the residue of by-products which are useless and which may become harmful. Elimination of these by-products, harmful or otherwise, occurs (1) through the biliary tract and out through the lower intestine, and (2) through the systemic circulation after detoxification through the urinary system.

Up to this point in the cycle, the blood contains eliminatory bodies which are derived solely from the breaking down of the food materials.

The systemic circulation represents the peak of production and distribution. The products of digestion are distributed throughout the body, and are utilized in the structural units of the tissues. In the process of utilization, catabolistic bodies appear which are destined for elimination from the body.

At this stage of the metabolic activity of the body, the systemic circulation contains two distinct sets of excretory bodies; the previously mentioned group, which is derived from and during the digestion of food, and a group resulting from the breaking down of the structural units of the tissues. As the materials for the structure of the food and of the tissue units are essentially the same—proteins, fats, carbohydrates, etc.—it is found that within certain restrictions each set of excretory bodies has a generally similar chemical structure. Both of these distinct groups mingle in the systemic circulation. From this point, elimination increases and becomes dominant.

A third set of excretory bodies is derived from the breaking down of the coloring matter of the blood. These bodies ultimately become the bile pigments and are excreted from the body economy into the bile.

The organs of elimination include the kidney, the intestinal tract, the skin and the pulmonary surface. The intestinal tract differs from the others in that some of the bodies excreted into its lumen for purposes of elimination are reabsorbed into the system to be handled and reexcreted again through the biliary tract into the intestine or to be passed out of the body through the kidney. Each of these avenues of elimination can under given circumstances attempt to assume the excretory functions of the others; the degree of compensation is, however,

of only relatively small degree, and in actual conditions of disease, insufficiency of the one is not compensated to the degree compatible with health by any or all of the others.

The accompanying diagram shows the interrelationships of the paths of absorption and elimination.

DIGESTIVE CHANGES

The chief changes which the foods undergo in the intestinal tract and by which they are made available to the body by be summarized briefly as

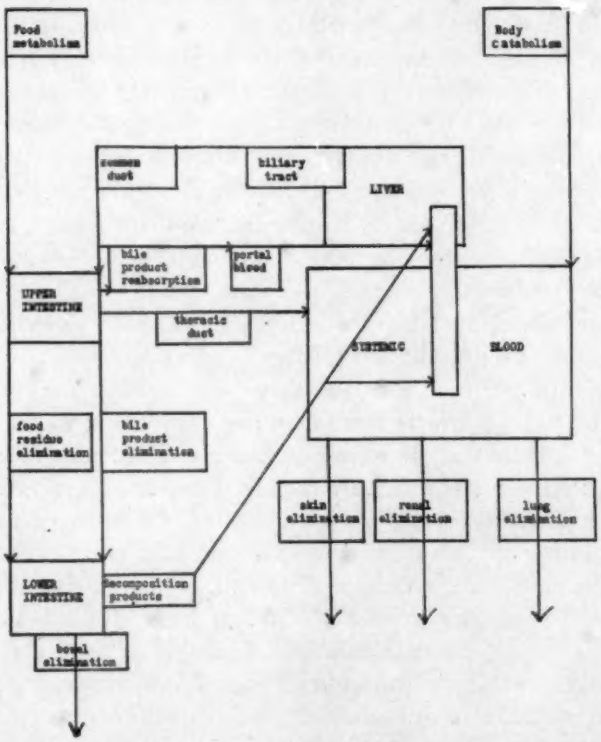

Fig. 1.—A graphic representation of the general relationships of the liver, the intestinal tract and the systemic circulation.

follows: The proteins by the action of pepsin hydrochloric acid of the gastric juice, by the intestinal and pancreatic juices containing enterokinase, trypsin and erepsin, are resolved into the amino-acids, of which they are composed. The compound proteins, such as the nucleoproteins, are digested in part by the proteolytic enzymes and in part by the nucleases of the pancreatic juice, with the formation of phosphoric acid, nuclein bases, pyrimidin bases and presumably carbohydrate, from the nucleic acid. Hematin is split off from hemoglobin. Some of these

amino-acids are further decomposed by the action of the bacteria losing ammonia and being converted into fatty acids, or by decarboxylization being made into amines, some of which have a powerful action on the blood pressure and other functions of the body. Other ill-smelling and harmful substances are produced, such as some of the mercaptans, sulphurated hydrogen, indol and scatol.

NITROGEN METABOLISM

Urea Formation.—In the process of digestion and absorption of protein food, large amounts of ammonia are set free. This ammonia is liberated in part as the result of the action of the enzymes of the digestive juices; in part, by the action of the bacteria in the intestine, and in part, during the process of absorption, from the amino-acids of the proteins of the food. The ammonia thus liberated accumulates in the mucous membrane of the stomach and intestine, whence it is gradually removed by the blood and carried to the liver in the portal blood. The liver converts at least part of it into urea, and the kidney eliminates it from the body. Another portion of ammonia is set free in the liver itself from the decomposition of the amino-acids brought to that organ in the portal blood, and the carbon moieties of those molecules are converted in part into glucose and glycogen. A relatively large fraction of the nitrogen of the food thus never becomes part of the living protein of the body, but is converted into urea and thrown out of the body. On a meat diet the amount of nitrogen thus converted into urea, so-called superfluous nitrogen, is relatively large. It is not certain just how large it is, but it probably is at least a third of all the nitrogen in the urine, since on fasting the urea drops at once to about two thirds its former value. Certainly with an output of 35 Gm. of urea a day, this digestive nitrogen will represent at least 10 Gm. of urea. This is not the only origin of the urea. The tissues are also capable of tearing their protein to pieces and deamidizing the amino-acids. For example, if a muscle has not a sufficient amount of carbohydrate to supply its energy, it will tear its protein to pieces to get the energy. This is always accompanied by a process of oxidation, during which process a ketonic acid is formed from the amino-acid and ammonia is set free. Lactic acid is formed in muscles during their work; some ammonia probably escapes from the muscles in the form of ammonium lactate, and this is in part made into urea. The endogenous urea has, therefore, a varied origin, coming from the different organs in the form of a variety of nitrogenous substances which are somewhere, most probably in the liver, made in part into urea. Even on the minimum protein diet of only 3 or 4 Gm. of nitrogen intake a day, the urea still makes 60 per cent or more of the total nitrogen in the urine.

That the liver in mammals is the chief organ producing urea from cleavage products of protein metabolism and from ammonia absorbed from the alimentary canal has long been held as probable, although not so definitely proved as to go unchallenged. Folin and his co-workers have reported experiments which they interpret as showing that in mammals, uric acid, for example, is destroyed principally in the blood; but the recent work of Mann and his associates by methods of total extirpation of the liver seems to show that the liver is the main organ that forms urea and destroys uric acid. Of course, even the experiments of Mann are open to the objection that the failure to produce urea and destroy uric acid after liver extirpation may be due to indirect impairment of other tissues or changes in the blood composition. At present the latter alternative seems the least probable one. Liver perfusion experiments, as well as portal and hepatic venous blood chemistry, seem to show a considerable demidization in the liver, but this is probably not an exclusive liver function. These processes probably go on to a certain extent in every living cell in the body. While the so-called meat poisoning following high protein diet in dogs with Eck fistula indicates a definite rôle of the liver in connection with taking care of some of the immediate end-products of the protein digestion or the regulation of cellular protein metabolism, neither blood nor urinary analysis in such meat intoxication has yielded the final answer as to the precise nature of these processes or even the specific substances responsible for the symptoms of intoxication. The intoxication does not appear to be due to ammonia.

Uric Acid Formation.—It has been proved that uric acid is derived from the metabolism of nucleoproteins (purine bases). It is believed that the greatest bulk of the uric acid excreted from the body comes from an exogenous source and is derived from the nucleoproteins of the food. In most tissues taken as foods, the bases in the nucleins have already been partially oxidized by the action of autodigestive and oxidizing enzymes of the tissues while still in the nuclein molecule, so that they are free in the intestine in a partially oxidized form as hypoxanthine, xanthine, guanine and adenine. These bodies are absorbed at least in part. Their absorption is followed by the appearance of some uric acid in the urine. The exact locus in which all of these changes occur is not exactly known, but it is worthy of note that of the total purine ingested only a small part appears in the urine as uric acid. The experiments of Bollman, Mann and Magath have given a great deal of information concerning uric acid metabolism.

According to Bollman, Mann and Magath, large amounts of uric acid appear in the urine of dogs following hepatectomy, which also gives rise to an increase in the uric acid content of the blood. Uric acid injected intravenously into animals following hepatectomy may be almost

quantitatively recovered in the urine. Uric acid injected into animals with the liver and both kidneys removed is slowly absorbed by the muscles, so that it does not all remain in the circulating blood. Determination of the uric acid content of the blood and muscles several hours after injection shows that the total amount is retained in the body and that little of it could have been destroyed. The uric acid accumulated in the body of hepatectomized neprotectomized dogs is of the same magnitude as that excreted by the kidneys of hepatectomized animals. The uric acid excreted by the hepatectomized animal is of the same magnitude as the allantoin excreted by the normal dog. The destruction of uric acid in the normal dog depends on the presence of the liver, as this process ceases as soon as the liver is removed.

Creatinine.—There is excreted in the urine of adults about 1 to 2 Gm. of creatinine a day. The amount is entirely independent of the protein intake, but is increased by the ingestion of creatinine, most of which reappears unchanged in the urine. The ingestion of creatine increases creatinine slightly, if at all; it may increase somewhat the creatine excretion. What becomes of the greater part of the creatine thus ingested is unknown. Creatine is found only occasionally in small quantities in the urine of men, a few milligrams a day, but it occurs, or is supposed to occur, in larger amounts, from 60 to 100 mg. a day in the urine of women during menstruation, in the first few days after childbirth and during pregnancy. It is usually present in children's urine, particularly in girls', and may comprise from one tenth to one third the amount of creatinine.

The amount of creatinine excreted in adults is roughly proportional to the body weight, from about 7 to 11 mg. of creatinine nitrogen per kilogram body weight being excreted in twenty-four hours. This figure is called the creatinine coefficient. The combined creatine and creatinine content undergoes little change during fasting, but the amount of creatinine diminishes and that of creatine increases, if the method of determination of creatine is correct. The same result occurs in carbohydrate starvation, in diabetes, both natural and phloridzin, and in the early stages of wasting diseases, such as fevers, muscle atrophy and other conditions. These facts show that creatinine has an endogenous origin, and that unlike urea it is unaffected by the intake of protein.

There is found in the voluntary muscles about from 0.35 to 48 per cent of creatine; large amounts are found also in the liver, the involuntary and heart muscle, in the brain, the testes and other organs. The total amount of creatine in the body is thus about 120 Gm. This creatine of the body is probably the origin of the creatine and creatinine of the urine. The muscles have the power of forming creatine from glycocyamine, and they and other organs can turn creatine to creatinine. Extracts of the organs have this same power, which is probably to be

ascribed to a ferment. Probably the kidneys are active in changing the creatine arriving by the blood to creatinine, but it is certain that they are not the only organs having this property. The tissues are also able to destroy creatine, but what is formed from it is unknown.

The increase of creatine in the urine in diabetes and carbohydrate starvation or after the injection of thyroid extract is ascribed by Folin, Shaffer and others to the increased catabolism of the tissues, but it is also possible that it is due to the setting free of the creatine from the combination in which it occurs in the muscles. Nothing definite is known of the function of creatine in the muscles and the other organs; whether it is primarily a waste product, or whether by its presence it affects the metabolism of the tissues, is not known. Further investigations are necessary. While all the really important questions about the origin and significance of creatine and creatinine still remain unanswered, it appears that these substances hold an important place in metabolism.

TABLE 1.—*Percentage of Urinary Output of Nitrogen Bodies Under Normal Conditions*

Exogenous Source (Food)	Urine Nitrogen, per Cent	Urine Nitrogen, per Cent	Endogenous Source (Tissue Catabolism)
Proteins-amino-acids-urea.....	60	30	Tissue (muscles); ammonia-urea
Nucleoprotein-uric acid.....	90-95	5-10	Gland origin; white blood cells-uric acid
Creatinine only under forced.....	(?)	100	Creatinine (creatine conversion)

The constancy of the excretion of creatinine indicates that it is, as Folin suggested, an index of the real catabolism of the vital machinery of the body proper, in distinction from that catabolism which increases the free energy.

URINE NITROGEN

Under ordinary circumstances, the urinary output of nitrogen bodies is found in the following proportions: Of the total amount of urea excreted in the urine, 60 per cent is derived from the breaking down of the proteins in the food, and 30 per cent is derived from the breaking down of the proteins of the body tissues, especially of the muscles. Ninety-five per cent of the uric acid is derived from the nucleoproteins of the food; 5 per cent is derived from the metabolism of the body tissues, especially its glandular organs. Under extraordinary conditions, a large excretion of uric acid which has been experimentally proved to follow the removal of the liver, is to be interpreted clinically to indicate destruction of liver tissue. All of the creatinine can for practical purposes be assumed to be derived from tissue breakdown; in exceptional instances, when the diet contains extraordinary amounts of creatine and creatinine, increased amounts of the later appear in the urine, being derived partially from this source.

BLOOD NITROGEN

Knowledge of blood chemistry is of comparatively recent origin. The work of Folin and Denis in the development of a simplified technic for the accurate estimation of blood urea probably gave the first great impetus to this investigation. Determinations by modern methods are feasible for any well equipped clinical laboratory and the technic of the procedure can readily be mastered. The development of simplified methods of estimating the amount of the various substances in health and disease has rapidly led to the accumulation of much data concerning their variations.

Blood Urea.—The normal urea content of the blood is subject to considerable variation. It is recognized as varying normally from 20 to 40 mg. per hundred cubic centimeters. In the interpretation of the content of the blood in every given case, the diet of the patient must be taken into consideration. It is at once apparent that a blood urea content of 50 per cent in a person on a nonprotein diet has not the same significance as the same level of blood urea in a person whose protein intake is high. Another factor which should receive consideration in the interpretation of the blood urea level is the fluid intake. It has been noted repeatedly that a high blood urea content can be reduced noticeably in many instances following the ingestion of large amounts of water. The amount of urea excreted can be shown to vary considerably with the amount of urine output. This is especially true when the renal damage is not extreme.

Other things being equal, a rise of blood urea above normal is primarily to be interpreted as an index of bilateral renal insufficiency; however, it is common experience that not infrequently patients can maintain a fair degree of health for a considerable period of time with a urea of 80 mg., and in occasional cases at a point considerably higher. On the other hand, there is occasionally definite uremia with a blood urea content within the normal range, so that an accurate prognosis cannot always be drawn from the urea content alone, as it is frequently necessary to take other factors into consideration.

When the blood content of urea rises to 100 mg. or higher, the prognosis becomes increasingly grave. An amount above this level can seldom be maintained for any considerable period without grave symptoms of renal insufficiency. In acute renal processes, in acute exacerbations of chronic renal disease or in urinary obstruction, the urea level may rise to an astonishingly high figure without a necessarily fatal termination, with subsequent recovery and a return of the urea to the normal level. Numerous instances of this kind have been reported frequently. These high figures are rarely attained in cases of chronic renal insufficiency, and when they occur nearly always terminate fatally.

Blood Nonprotein Nitrogen.—Estimations of nonprotein nitrogen are utilized in some laboratories instead of estimations of blood urea. The technic for the estimation of nonprotein nitrogen is considerably more complicated than is that for blood urea. The normal blood content of nonprotein nitrogen varies approximately from 30 to 50 mg. per 100 cc. of blood. Definite figures are especially difficult to establish. Data regarding the nonprotein nitrogenous blood components have attained undeniable value in the interpretation of kidney function. They are dwelt on in the clinical laboratory of today as indexes of the degree and type of retention occurring in specific cases, and they influence both the diagnosis and the prognosis. The details of the distribution of the nonprotein nitrogenous components in the blood have been studied with respect to their bearing on nitrogen metabolism, as well as the degree of elimination of nitrogenous waste.

The accumulated data have been somewhat disappointing to many persons, notably to clinicians. They have been encouraged by the information secured from blood chemistry in respect to renal functions and the corresponding conditions of the kidney. They had come to expect that further data on noprotein nitrogen in cases uncomplicated by adverse kidney factors might throw light on obscure diseases, particularly those having a background of metabolic disorder. Numerous laborious collections of statistics regarding the nitrogenous composition of the blood in diseases of the nervous system have failed to elucidate the pathogenesis or the pathology of the subject.

Blood Uric Acid.—The normal blood content of uric acid reflects the nucleoprotein content of the food from which it is normally almost entirely derived, and ranges between 1.5 and 3.6 mg. per 100 cc. of blood. In certain physiologic periods, notably the later months of pregnancy, the uric acid content of the blood rises considerably; this is a physiologic phenomenon related to pregnancy. Experimentally as referred to previously, the removal of the liver (Bollman, Mann and Magath) is followed by the accumulation of uric acid in the blood stream. A similar increase in the uric acid of the blood has been noted by Lenox to follow prolonged starvation. The exact mechanisms causing these increases is not definitely known.

Uric acid circulates in fairly constant concentration in the blood of men, any increment in the amount being indicative of a pathologic condition. Whatever the intermediate reactions may be, an increased output of uric acid is demonstrably an indication of an increased transformation of purine yielding products, whether the latter are derived from the diet or have an endogenous origin in the cells and tissues of the body. Deductions from the work of Bollman, Mann and Magath, his co-workers, referred to previously, seem to show that in liver conditions high concentrations of blood uric acid can be used as a differential point as indicating liver cell destruction.

Blood Creatinine.—Quantitative determinations of blood creatinine give information concerning one of the end-products of endogenous metabolism. The amount of creatinine in the blood is not influenced, as is urea, by the protein intake, so that it would be expected that a rise of creatinine above normal would be a truer index of tissue metabolism. The normal blood content of creatinine averages about 2 mg per 100 cc., and as a rule, there is no appreciable increase in this amount until well marked renal insufficiency is present, as determined by blood urea and phenolsulphonphthalein estimations.

When there is a marked increase of creatinine, 5 mg or more, a serious disturbance of renal function occurs. The combination of a high urea content of the blood with a high creatinine content of the blood offers a distinctly graver prognosis than the same amount of urea with a normal creatinine content. It is at once apparent that the value of this determination as a functional test is limited and is by no means as delicate an index of renal function as the estimation of blood urea; furthermore, it seldom gives information which is not obtained by the determination of urea. It has been found by Squier and Myers, and by Frontz and Geraghty, that creatinine is much more readily excreted than urea and uric acid, and that considerable diminution in renal function can occur before there is any rise in the creatinine level.

Amino-Acids.—The hope of gaining unusually valuable facts from the contemplation of the amino-acid content of the blood has also not been realized. Folin has pointed out that this is what one might be led to expect. Deamination must be a prelude to the destructive utilization of much of the protein material either ingested or liberated from tissue sources. The long debated theory that deamination precedes in the walls of the intestine has been abandoned. One might readily expect anomalies of deamination to occur as manifestations of metabolic disorder; indeed, the well known failure of cystine to be consumed in the organism of certain persons furnishes an illustration of this type of defect. But, with the exception of rare instances of such inborn errors of metabolism, deamination abnormalities appear to be of rare occurrence. Folin has remarked that the deamination process appears to be such a fundamental process that one cannot expect to find many pathologic conditions in which the amino-nitrogen of the blood filtrates will vary much from the normal.

The amino-acid content of the blood represents a food product on its way from the intestine to the tissues. The amount of amino-nitrogen in the blood varies between 4.8 and 7.8 mg. in 100 cc., the average amount being 6.3 mg. This was found by Greene, Sandiford and Ross to be true in normal persons, and in a series of more than 400 observations covering twenty pathologic conditions. A definite increase in the amino-nitrogen content of the blood during protein digestion was first

TABLE 2.—*Cases with Mechanical Defect (Hernia) but Otherwise in an Apparently Normal State of Health*

Case	Urea Nitrogen	Nonprotein Nitrogen	Uric Acid	Creatinine
1	12.6	27.3	3.5	1.0
2	17.6	33.3	3.5	1.1
3	18.2	35.0	3.4	1.1
4	14.0	30.0	2.5	1.1
5	....	34.1	2.5	1.0
6	11.2	....	3.5	1.0
7	18.2	34.1	1.7	1.0
8	15.4	33.3	3.3	1.2
9	14.0	....	2.3	1.2

TABLE 3.—*Cases with Mechanical Defect (Postoperative Adhesions) with Various Grades of Symptoms but Without Signs of Interference with the Movement of the Intestinal Content—Without Nephritis and in Otherwise Good Physical Condition.*

Case	Urea Nitrogen	Nonprotein Nitrogen	Uric Acid	Creatinine
1	14.0	33.3	4.0	1.2
2	12.6	34.1	2.0	1.1
3	12.6	29.3	1.6	1.2
4	11.2	....	3.0	1.1
5	12.5	21.3	...	0.75
6	21.25	42.5	...	1.25

TABLE 4.—*Cases in a Physiological Period (Pregnancy) Occurring Normally and Without Abnormal Symptoms*

Case	Month	Urea Nitrogen	Nonprotein Nitrogen	Uric Acid	Creatinine
39	6	11.0	21.0	1.1	1.1
40	8	8.5	22.5	2.2	1.2
41	3	10.0	23.0	1.3	1.1
42	8	10.0	24.0	2.5	1.3
43	5	11.0	25.0	1.9	1.3
44	8	9.5	23.7	2.3	1.3
45	7	17.0	25.0	1.9	1.3
46	8	11.0	15.0	2.2	1.3
47	4	9.0	25.0	Trace	1.3
48	9	15.0	28.0	Trace	1.6
49	6	10.0	25.0	2.3	1.3
50	3	10.0	22.5	2.3	1.1
51	7	12.0	20.0	2.2	1.2
52	7	10.7	22.5	2.6	1.5

TABLE 5.—*Chronic Diseases (Malignancy, etc.) in Elderly People Without Manifestations Referable to any Change or Disturbance of Metabolism*

Case	Diagnosis	Urea Nitrogen	Nonprotein Nitrogen	Uric Acid	Creatinine
1	Gangrene of foot	16.8	33.3	1.5	1.7
2	Gangrene of foot	11.2	30.7	2.5	1.1
3	Carcinoma of stomach	13.6	34.1	2.5	1.0
4	Calcified abdominal nodes	21.0	42.0	4.0	1.2
5	Retroperitoneal tumor	28.8	33.3	2.5	1.0
6	Posthysterectomy abdominal pains	14.0	33.6	2.0	3.5

conclusively demonstrated by Van Slyke, who utilized his own nitrous acid method. Subsequent investigations have shown the possibility of following the metabolism of these bodies, their storage in the liver and muscles and their ultimate deamination in the latter organ. Apart, however, from the great increase in the amino-nitrogen content of the blood in acute yellow atrophy—an increase which is in part attributable to the massive tissue autolysis which is occurring, and in part to the failure of deamination resulting from destruction of the liver tissue—the estimation of these bodies is at present of little importance in medical practice.

CLINICAL FACTS

Indications of all of the metabolic changes referred to in the previous part of this article, whether they be derived from ingestion or from tissue breakdown, are distinguishable in the actual and relative proportions of all of these important bodies as they are being transported in the blood stream. However, when an attempt is made to correlate the relative and proportional status of these bodies at any given moment with conditions of health and disease, it is found that constancy does not exist; seemingly, there is an extraordinary amount of confusion; and this seems especially to be the case in the correlation of the blood figures with pathologic states. The wide extent of the "normal" variation is another source of difficulty. The observations in various conditions are of great interest in this regard. These are shown in tables 2 to 5.

Summary.—The chemical composition of the peripheral blood depends on the chemical composition of the cellular elements of the blood and on the chemical composition of the blood serum. The first of these is a constant factor. The second of these, the blood serum, is a constantly varying combination of numerous constituents which can roughly be divided into a group of bodies that are on their way to being built into established tissue elements, that is, the amino-acids; a group of bodies which are derived from tissue catabolism and represent structural wear and tear; a group of bodies similar to those in the previous two groups, which are derived from the ingested food during digestion and represent the wastage of an excess food supply, and a series of bodies due to the action of intestinal bacteria on the digestion residues in the alimentary canal. At any given moment the relative proportions of all of these varying groups of bodies existing in the blood serum represents a balance between the withdrawals and additions from and to the blood stream.

Exaggerations of all of these variations accompany certain physiologic epochs (1) in the daily life of any person, that is, the taking of food, periods of exercise, periods of sleep; and (2) in the life cycle, that is, pregnancy. Extreme variation occurs between persons between successive periods of life, and with certain underlying manifestations which one is accustomed to designate as "diathesis."

The faculty for making use of various materials for producing the same end-product gives rise to compensatory powers which frequently keep the blood index at normal levels when pathologic conditions are present in which one is accustomed to find differences in the blood indexes.

Multiplicity of methods and mechanisms whereby similar metabolic changes can occur in different regions of the body give ample opportunity for wide compensatory powers; wide reserve powers are thereby created; all of these give opportunity for additional variation.

It seems correct to assume from these observations that in any given case a normal must be established for each person at the moment of examination. Single observations are of little moment; a series of observations are necessary. Comparisons of persons and of disease can be made only from comparable series of observations taken under normal and abnormal conditions; actual figures mean less than similar plus or minus changes viewed in the light of repeated observations and of actual pathologic conditions and controlled by a knowledge of the underlying opposing factors the resultants of which form the balance to be found in the blood stream. Abnormalities are to be determined only along these lines and according to these criteria.

TORSION OF THE GALLBLADDER*

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BALTIMORE

Torsion of the gallbladder is a rare condition. I have been able to find only two American reports, that of Wendel in the *Annals of Surgery* for 1898, and that of Cramp in the *Medical Record* for Jan. 16, 1915.

Most of the cases reported have been published in the British and German journals. In Kehr's *"Chirurgie der Gallenwege,"* published in 1913, five cases of complete torsion are mentioned: those of Mühsam, Nehrkorn, Mayer, Fischer and Kubig. He evidently had not looked outside the German literature.

It is necessary to distinguish between partial and complete torsion. Sutter reports one case of complete torsion and two cases of incomplete torsion. The reports of Aladar Fischer, Asteriades and Seefisch discuss incomplete torsion, with which I am not directly concerned in this article. Clinically, there is a sharp distinction between the two types. In complete torsion there is strangulation of the blood supply and gangrene of the gallbladder, and the symptoms begin with dramatic suddenness. In incomplete torsion the symptoms are due to interference with gallbladder drainage, and more closely resemble those of chronic cholecystitis.

Angulation of the cystic duct is a separate condition. Krukenberg reported two such cases in 1913. He described the free gallbladder with a short mesentery and named this condition "wandering gallbladder."

The anatomic relationship of the gallbladder to the liver varies widely. Roughly, this relationship may be considered in four phases, as described here.

1. In rare cases the gallbladder is found entirely imbedded within the liver, and its surface is completely hidden. The cystic duct, however, is usually outside of the liver, and the fundus of the gallbladder is felt as a soft area in the liver, just above its anterior margin.

2. In many gallbladders a large portion of this viscus is covered by peritoneum, but some portion of the wall of the gallbladder throughout most of its length adheres to the liver, with no tissues interposed. When such a gallbladder is removed, it leaves a considerable area of denuded liver, to which some abdominal structure is likely to adhere.

3. In many cases, the gallbladder is attached to the liver throughout its length, but nowhere touches it closely. Between the gallbladder and liver a layer of fibrous tissue is interposed, forming a sort of mesentery.

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*From the Surgical Service of the University of Maryland Hospital.

The blood vessels run in this layer, and it is covered in front and behind by the peritoneum, which is reflected over it from the gallbladder to the liver. Such a gallbladder is usually freed with little difficulty from the liver and leaves a relatively narrow area of denudation, which is easily covered.

4. The last group includes uncommon cases in which the entire gallbladder is covered by peritoneum, and is free. The gallbladder is attached to the liver by a short mesentery which extends along the cystic duct and usually terminates near the point where the cystic duct joins the gallbladder. In this mesentery are the cystic vessels. It is in cases of this kind that torsion of the gallbladder occurs. There is considerable medical literature calling attention to this condition.

In 100 autopsies, Brewer found that the gallbladder had a mesentery-like attachment in five cases. In another report Ssuslow mentions six similar mesenteries in 145 persons examined. Jacquement found it twice in 200 patients; Raynal twice in thirty-six.

I have found in the literature twenty-one cases of complete torsion of the gallbladder, to which is added the case of my patient. A brief synopsis of these cases follows.

REPORT OF CASES

CASE 1.[1]—A woman, aged 23, had been under observation from November, 1895, until March, 1897. In November, 1895, a mobile ovoidal tumor was found in the upper right quadrant. The tumor was about 2 inches (5.08 cm.) long and 3 inches (7.6 cm.) wide, and was cystic in nature. The right kidney was loose, and could be moved as far as the umbilicus. The patient refused to submit to an operation. She was treated, and declared herself well until Sept. 20, 1896, when she was suddenly seized with severe pain in the upper part of the right side of the abdomen, with vomiting, high fever and abdominal distention. At this time the cystic swelling could not be felt. There was a fixed, rigid, painful swelling in its position. The woman again refused to submit to an operation and soon recovered from the attack. In the early part of March, 1897, she returned to the hospital, saying that she was two months pregnant. On March 25, 1897, she was operated on. At operation, perforated cholecystitis, cholelithiasis and a peritoneal abscess were found. Two hundred and thirteen gallstones and the gallbladder were removed. The gallbladder was not gangrenous, but was much twisted on the cystic duct. It was adherent, but the adhesions were recent. The cystic duct was 3¼ inches (8 cm.) long, with a mesentery about 2 inches (5 cm.) long, attached to the inferior surface of the liver. The neck of the gallbladder was obstructed by the largest stone. The patient recovered.

CASE 2.[2]—A woman, aged 54, became ill two days before coming to the hospital, with a sharp pain in the abdomen. The condition was not accompanied by vomiting. When she was admitted the abdomen was rigid and her pulse

1. Wendel, A. V.: A Case of Floating Gall Bladder and Kidney Complicated by Cholelithiasis with Perforation of the Gall Bladder, Ann. Surg. **27**:199, 1898.

2. Mayer, W.: Ein weiterer Fall von Stieltorsion der Gallenblase, Berl. klin. Wchnschr. **14**:1602, 1908.

rate was 150. There was an inguinal hernia on the right side. Directly above the hernia a sausage-shaped tumor the size of an infant's head was felt. The patient was in a state of collapse. A diagnosis of intestinal obstruction was made. Operation disclosed a large black mass, which was thought at first, to be hydronephrosis in a wandering kidney. Further examination proved that the swelling was an enlarged and strangulated gallbladder, which was rotated twice on the cystic duct. There were four stones in the gallbladder, the walls of which were thickened and infiltrated with blood. The cavity of the gallbladder was also filled with blood. Cholecystectomy was performed. The patient collapsed on the table and died the following night.

CASE 3.[3]—A woman, aged 74, had suffered from attacks of sharp abdominal pain from the time she was a young girl, but she had become well in her old age. Eight days before admission to the hospital she had a sudden attack of pain in the upper part of the right side of the abdomen and in the back. Her bowels ceased to move, and she vomited. There was a great deal of resistance in the muscles of the upper part of the right side of the abdomen. Masses could not be felt. The condition was thought to be empyema of the gallbladder. When the abdomen was opened, a dark gallbladder was seen, surrounded by fine adhesions. When these adhesions were separated, torsion of the gallbladder on the cystic duct was found. There was a short band of adhesions running from the cystic duct to the liver. The gallbladder was entirely surrounded by peritoneum and was not perforated. Cholecystectomy was performed and the patient recovered.

CASE 4.[4]—A woman, aged 61, had an attack which began with a slight pain in the upper part of the right side of the abdomen. The bowels were regular, and there was no vomiting. The attack, which began four days before admission to the hospital, later became more severe, and there was some vomiting. There was some pain in the right shoulder. The leukocyte count was 20,000 on admission. There was marked spasm of the abdominal muscles, especially on the right side. There was great tenderness and a mass in the gallbladder region. Jaundice was not a symptom. The heart, lungs and kidneys were normal. The diagnosis was either appendicitis or gallbladder disease. When the patient was put under anesthesia, a definite pear-shaped tumor was felt, extending from the region of the umbilicus into the region of the gall-bladder. The diagnosis was then empyema of the gallbladder. When the abdomen was opened, a moderate amount of bloody serum escaped, and the gallbladder, a dark red, sausage-shaped mass, the size of a goose egg, was found to be completely twisted from right to left on the cystic duct. It was removed. The walls were infiltrated with blood, and one stone was found in it. The patient recovered.

CASE 5.[5]—A woman, aged 72, had a sudden attack of upper right abdominal pain three days before admission. She had not been ill previously. Jaundice was not a symptom. A round tumor could be felt in the region of the gallbladder. The condition was diagnosed as acute cholecystitis. Operation showed a gallbladder about the size of a pear. It was gangrenous and sur-

3. Nehrkorn: Gangrän der Gallenblase durch Stieldrehung, Deutsche Ztschr. f. Chir. **96**:319, 1908.

4. Mühsam, R.: Stieldrehung der Gallenblase, Berl. klin. Wchnschr. **14**: 1179, 1908.

5. Lett, Hugh: Two Unusual Conditions of the Gall Bladder. One a Case of Torsion, Lancet **1**:1099 (April) 1909.

rounded by recent adhesions, which were separated with the finger. There was complete axial rotation of the gallbladder on the cystic duct from left to right (counter-clockwise). The gallbladder was free, with a short mesentery. Gallstones were not present. Cholecystectomy was performed, and the patient died twelve hours later.

CASE 6.[6]—A woman, aged 70, two days before she came to the hospital, had an attack which began with pain in the abdomen, like that of acute appendicitis. She vomited and there was an elevation of temperature. Her tongue was dry and coated, and her whole abdomen was tender. Tenderness and rigidity were most marked in the right upper quadrant. A diagnosis of acute appendicitis with abscess or cholecystitis was made. When the abdomen was opened, a considerable quantity of bloody serum escaped. The appendix was normal. A large, bluish mass, the size of a goose egg, was found to be the gallbladder. It was attached to the liver only by a twisted pedicle and was covered with a grayish exudate. The twist was 360 degrees (counter-clockwise), and was entirely on the cystic duct. Stones could not be felt in the common duct. Bloody serum was present. The gallbladder was not ruptured. It was removed, and the patient recovered.

CASE 7.[7]—This was a case of volvulus of the gallbladder found at autopsy in the body of a man 73 years old. During his last illness, he had a severe cough. He died in the Tuberculosis Pavillion. At autopsy profuse bronchitis and hyperemia of the lungs, bronchopneumonia and torsion of the gallbladder with circumscribed peritonitis were found. The gallbladder was twisted clockwise on the cystic duct 270 degrees, was dark blue and was free. There were no gallstones, and the gallbladder was not gangrenous or thickened. It was entirely covered with peritoneum.

CASE 8.[8]—At a meeting of the New York Academy of Medicine in the Section on Surgery, held Dec. 4, 1914, Dr. Cramp presented a patient, who had been admitted to Bellevue Hospital suffering from pain in the abdomen, nausea and vomiting. This patient was a man whose age was not given. From the history of the case it was supposed that his trouble was due to indiscretion in diet, and he was placed in the medical division. A tentative diagnosis of gastritis was made. The following day he was transferred to the surgical division. There was rigidity over the right upper quadrant and a temperature of 99 F., but the symptoms did not indicate obstruction. There was a high leukocyte and polymorphonuclear count, and an epigastric tumor. Immediately after the incision was made, the gallbladder protruded through the opening. It was black and twisted one and one-half times from right to left on its pedicle. It did not communicate with the liver, except toward the common duct. Dr. Cramp presented the specimen removed, which did not resemble a gallbladder whatever. Cholecystectomy was performed, and the patient recovered.

CASE 9.[9]—A woman, aged 71, was taken suddenly ill three days before admission to the hospital. She had not had a previous attack. The first symptom

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6. Fischer, August: Ein weiterer Fall von Stieltorsion der Gallenblase, Berl. klin. Wchnschr. **18**:1784, 1910.

7. Kubig, G.: Ueber Volvulus der Gallenblase, München. med. Wchnschr. **9**:1998, 1912.

8. Cramp, Walter C.: Gangrene of Gall Bladder from Twisted Cystic Duct, Med. Rec. **87**:120 (Jan. 16) 1915.

9. Reichel: Volvulus der Gallenblase, München med. Wchnschr. **3**:884 (Aug. 1) 1919.

was pain; there was a stool the evening she became ill. After this there was no movement, and there was eructation of gas and vomiting. On admission the patient's general condition was good, the pulse rate was 88. The abdomen was distended. There was pain and resistance on the right side of the abdomen; a mass could not be felt. The diagnosis was acute appendicitis. The appendix was exposed and found normal. Some fluid was seen, and the incision was enlarged upward and a gangrenous gallbladder, attached to surrounding structures by fine adhesions, was exposed. The gallbladder was twisted on the cystic duct 360 degrees from right to left (clockwise). Cholecystectomy was performed, and there was a small drain. The patient recovered.

Case 10.[10]—A woman, aged 73, became ill four days before admission to the hospital with pain and constipation. When admitted she did not have fever, the abdomen was distended and a tender mass was felt under the liver. The diagnosis was ileus, thought to be due to cancer of the hepatic flexure. At operation, a gangrenous gallbladder was found twisted on the cystic duct 360 degrees. It contained one stone. Cholecystectomy was performed, and the patient recovered.

Case 11.[10]—A woman, aged 64, gave a history of repeated attacks of gallstone colic. An attack began the day before admission with severe abdominal pain, vomiting and constipation. She was in poor general condition, with abdominal distention. Under the liver a tender swelling the size of an apple was felt. The diagnosis was volvulus of the gallbladder. At operation, a large gangrenous gallbladder was found, twisted 360 degrees on the cystic duct. After cholecystectomy was performed, she recovered.

Case 12.[10]—A woman, aged 78, gave a history of short attacks of abdominal pain for two years. The onset of this attack was four days before admission. She suffered sharp pain in the entire abdomen, more marked on the right side. Constipation and vomiting were symptoms. Under the margin of the liver there was indefinite tender resistance. Operation disclosed a dark red gallbladder with 180 degrees torsion on the cystic duct. It contained one stone. Cholecystectomy was performed, and the patient recovered.

Case 13.[11]—A woman, aged 79, was suffering with severe pains in the abdomen. The past history was negative, except for appendicitis many years before. The onset occurred twenty-four hours before admission to the hospital. Terrific pains in the abdomen and nausea and vomiting were the symptoms. The vomitus was foul smelling and green. From that point on, she had no bowel movement and no escape of gas. The clinical diagnosis was ileus. Her temperature was normal. The pulse rate was 70 and the pulse beat was light and of good volume. The abdomen was large and flabby. Peristalsis was not visible. In the region of the gallbladder, one could feel a round, smooth, tense and tender mass, which extended three or four finger-breadths below the costal margin. There was a slight flatness on percussion, continuous with dulness in the liver. The rest of the abdomen was not tender, but was soft and flabby. The urine showed a trace of sugar, which disappeared after the operation. After an enema the patient passed some foul-smelling gas, but the foul-smelling, almost fecal-like vomiting continued. Cholecystitis with a stone in the cystic duct was considered as a diagnosis, but there were symptoms of ileus, and

10. Krabbel, Max: Die Stieltorsion der Gallenblase, Deutsche Ztschr. f. Chir. **154**:76, 1920.

11. Hansen, P. N.: Et tilfaelde af volvulus af galdeblaeren, Hospitalstid. March 30, 1921, p. 23.

this led to immediate operation. As soon as the peritoneum was opened, a thin, serous, bloody fluid exuded. A mass, which resembled an incarcerated or tortuous gangrenous intestine, appeared, but it was seen at once that it was not related to the intestine. The pedicle of the gallbladder was rotated 360 degrees clockwise. The pedicle was 3 cm. or 4 cm. wide, was thin and contained few veins, but nothing else was clearly defined. The pedicle stretched along the edge of the liver, and the liver was at that point atrophic, with some thickening of the serosa. The rest of the liver was irregular, with deep scars. The gallbladder and cystic duct did not contain stones. The gallbladder was directly continuous with the thick serous membrane of the gallbladder. The gallbladder and cystic duct did not contain stones. The gallbladder was divided into two parts by a stricture, and it was shaped like an hour-glass. Only the fundal part was strangulated. The fundus had definite motility, and this portion was free without mesentery. The patient recovered.

Case 14.[12]—A woman, aged 42, had been examined and treated for stomach trouble. The onset of her illness was sudden, with severe colicky pains in the abdomen, constipation and vomiting. She did not have fever. Under the influence of morphine and hot dressings the pain subsided, and a mass was felt on the right side, extending from the region of McBurney's point to the epigastrium. Examination was difficult, because the patient was fat and had a thick abdominal wall. She did not have jaundice. Her pulse rate was 108, and her temperature 38.2 C. The preoperative diagnosis was acute cholecystitis with hydrops. When the abdomen was opened, some serum was found. The gallbladder was surrounded by recent adhesions and was seen as a large mass. There was a band of adhesions running from the upper surface of the gallbladder toward the stomach and colon. This was cut between ligatures. The gallbladder was easily lifted from its bed and was free, except that it was attached to the liver by the cystic duct, on which it was twisted 180 degrees. The gallbladder was dark red and contained one large stone. Cholesystectomy was performed, and the patient recovered.

Case 15.[13]—A woman, aged 34, had had attacks of biliousness since girlhood. The onset of the present illness was acute. She did not have jaundice, distention or visible peristalsis. Masses could not be felt. Tenderness and muscle spasms were observed. At operation, a gangrenous gallbladder was found about the size of a small hen's egg. There was one complete revolution (clockwise). Stones were not found and mesentery and peritoneal covering were not reported on. Cholecystectomy was followed by complete recovery.

Case 16.[14]—A woman, aged 62, suffered a sudden colicky pain in the upper part of the abdomen. The right part of the abdomen was rigid, and a tumor could not be felt. At operation, the gallbladder was found to have rotated completely from right to left. It was free, with a short mesentery. It did not contain stones. Cholecystectomy was performed and the recovery was uneventful.

Case 17.[15]—A woman, aged 67, suddenly became ill. A sausage-shaped tumor was felt in the region of the gallbladder. At operation, a reddish-black

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12. Strauss, M.: Die Stielderhung der Gallenblase, Beitr. z. klin. Chir. **122:** 322, 1921.

13. Irwin, S. T.: Torsion of the Gall Bladder, Brit. J. Surg. **9:**310 (Oct.) 1921.

14. Frankau, C. H. S.: Torsion of the Gall Bladder, Brit. J. Surg. **10:**301 (Oct.) 1922.

15. Jonas, H. C.: A Case of Torsion of the Gall Bladder, Brit. M. J. **1:**1016, 1923.

mass was found; this was a gangrenous gallbladder. There was some fluid in the peritoneal cavity. The torsion was from left to right and ten or twelve moderately large gallstones were found. Cholecystectomy was performed, and the recovery was uneventful.

CASE 18.[16]—A case of torsion of the gallbladder in an old woman, which was diagnosed cholelithiasis, is reported. In the region of the gallbladder there was a large pear-shaped tumor. The attack came on sharply with pain. At operation an enormous, movable and elongated gallbladder with a narrow mesentery was found. The gallbladder was twisted on the cystic duct. Cholecystectomy was performed and the patient recovered.

CASE 19.[17]—A woman, aged 74, had frequent attacks of abdominal pain for a year preceding the operation. On Aug. 20, 1924, following a hard day's work in the field, she had sudden violent abdominal pain accompanied by vomiting. During the following days there was some improvement, then a new attack of pain with vomiting. On August 26, six days later, the patient was transferred to the hospital. In the upper right quadrant a sausage-shaped tumor was felt. Jaundice was not a symptom. This tumor was smooth and mobile, and was the size of a fist. At operation, a bluish-red, tight, spastic swelling was seen. It was a gangrenous gallbladder, twisted on its pedicle 360 degrees (counter-clockwise) and attached to the liver by a pedicle. When it was removed, an elongated pedicle with a cystic area was found buried in adhesions and was removed with difficulty; it was thought to be a distended cystic duct. Rapidly developing jaundice followed the operation, and the patient died twelve days later. At autopsy it was found that the pull on the gallbladder had elongated the common and hepatic ducts, and these had been ligated when the gallbladder was removed.

CASE 20.[18]—A woman, aged 54, had a sudden attack of severe pain in the upper part of the right side of the abdomen with frequent vomiting. It was the first attack. Jaundice was not present. Tenderness and rigidity in the upper part of the right side of the abdomen were pronounced, and a round lump suggestive of an enlarged gallbladder, could be felt. The preoperative diagnosis was acute cholecystitis with a stone impacted in Hartmann's pouch. At operation, some serous fluid escaped when the abdomen was opened, and a black distended gallbladder was presented. It was completely covered by peritoneum and attached by its proximal end to the liver by a mesentery, which contained the cystic duct and the blood vessels. A peritoneal fold ran from the cystic duct to the duodenum. There was one complete revolution of the gallbladder from right to left. The gallbladder contained bile mixed with blood, and its walls were much thickened by hemorrhagic infiltration, but it did not contain stones.

CASE 21.[19]—Marinacci reports a patient with a tumor mass in the position of the gallbladder. The tumor was rather low. A twisted ovarian cyst was suspected on account of the sudden appearance of the symptoms. Operation revealed an enlarged, distended gallbladder with very little liver attachment. The gallbladder was twisted on the cystic duct. A portion of the gallbladder

16. Dubs, J.: Ein Fall von Torsion der Gallenblase, Schweiz. Med. Wchnschr. **3**:93 (Jan. 17) 1924.

17. Sutter, A.: Torsion of Pedicle of the Gall Bladder, Beitr. z. klin. Chir. **133**:519-532 (March) 1925.

18. Fifield, L. R.: Torsion of Gall Bladder, Brit. M. J. **1**:920, 1925.

19. Marinacci, Sertorio: Torsion of the Gall Bladder, Acute Dropsy, Policlinico **33**:194 (Feb. 8) 1926.

was black and necrotic and nearly ready to perforate. The cystic duct was ligated, cholecystectomy was performed, and the patient recovered.

CASE 22.—A white woman, aged 65, was referred to me by Dr. E. M. Bush of Hampstead, Md. She was tall and thin, with a narrow chest and considerable splanchnoptosis. She was married and had seven children living and well. All pregnancies had been normal. She had not had previous symptoms of gastro-intestinal disease. Two days before admission, there was a sudden attack of severe, continuous pain in the upper part of the abdomen, beginning in the epigastrium and radiating transversely to the right. Nausea, vomiting, distention and jaundice were not symptoms. The bowels had been moving regularly.

On admission, the patient looked ill. Her pulse rate was 100 and her temperature was 100. The entire right side of the abdomen was rigid, although it was not distended. The point of greatest tenderness was midway between the costal margin and McBurney's point. The rigidity was so great that a mass could not be made out. The diagnosis was uncertain as between acute cholecystitis and acute appendicitis.

A right rectus incision was made and as soon as the peritoneum was opened, a considerable quantity of bloody serum escaped. A large tumor mass was found, which was a distended gallbladder, smooth, tight and black. There were no adhesions about it, and it was lying low in the abdomen because of a displaced liver, due to splanchnoptosis. When the intestines were packed off and the gallbladder delivered, a complete torsion of the cystic duct counter-clockwise was found. The gallbladder was entirely free and completely covered with peritoneum. It was attached to the under surface of the liver by a pedicle, which contained the cystic duct, and by a narrow mesentery, which contained the vessels. The twist was easily unwound, the cystic duct was severed between clamps, and the gallbladder was removed. The severed duct was ligated, and the subhepatic space drained. The abdomen was closed around these drains. Stones were not found. The gallbladder was black and was filled with blood. Its wall was thickened and infiltrated with blood. The gallbladder was not perforated, and peritonitis did not develop.

The patient made an uneventful recovery.

COMMENT

It is an outstanding fact that in none of these cases, except Wendel's, was the gallbladder perforated, although in two cases eight days and six days, respectively, had elapsed between the beginning of the attack and operation. In Wendel's case the gallbladder was perforated, but was not gangrenous and 213 stones were found. This patient, aged 23, was the youngest person in the series, and this was the only case in which there was a clear history of previous attacks of a similar nature. It is likely that the torsion in this case was incomplete and played only a minor part in the pathologic condition.

In Hansen's case there was an hour-glass gallbladder, and only the part distal to the constriction was gangrenous.

In one case, Sutter's, a cystic mass was found proximal to the gallbladder. This was thought to be the dilated cystic duct. It was dissected free and ligated. This patient had not had jaundice previous to the

operation, but developed a deep jaundice afterward. At autopsy it was seen that the pull of the gallbladder had elongated the common and hepatic ducts, and these structures had been included in the ligature.

All the patients were women, except two. Kubig's case occurred in a man, aged 73. This man had been a patient in the Tuberculosis Pavillion, and the condition of the gallbladder was not suspected before the autopsy. The other man was Cramp's patient. The evidence is conclusive that this condition is usually seen in older women, who are thin and relaxed, and who belong to the narrow, long-bodied type with more or less downward displacement of the abdominal organs. It would be interesting to find out the percentage of free gallbladders in persons of this type.

The gallbladder exhibited different degrees of necrosis in twenty cases, the only exceptions being Wendel's and Kubig's patients, in whom the torsion was somewhat less than 360 degrees. Both of these cases might be omitted from the list of complete torsion of the gallbladder.

The direction of the volvulus is apparently not a matter of significance; of the twenty-two cases, seven were clockwise, five were counter-clockwise, and in ten cases the direction was not mentioned.

In all cases of the series, except those of Kubig and Marinacci, the onset was sudden, with pain in the upper part of the right side of the abdomen as the outstanding symptom. Vomiting was fairly constant, so much so that in three cases ileus was suspected.

In these cases of torsion of the gallbladder most of the surgeons have reported the presence of a moderate amount of bloody serous fluid in the peritoneal cavity. This was true of my own case.

In almost every instance the gallbladder lay free in its bed. In a number of cases there were loose, thin, recent adhesions, but the delivery and removal of the gallbladder was uniformly not difficult.

There was little evidence of peritonitis. The outstanding pathologic condition was necrosis due to strangulation of the blood supply, and the only peritoneal involvements were the loose adhesions and the small quantity of bloody serum.

In the reports of this series of cases it was repeatedly mentioned that examination of the gallbladder showed its walls to be thickened and infiltrated with blood, and the cavity of the gallbladder was usually filled with blood also. The gallbladder was nearly always enlarged, was distended, smooth and elastic, and, in a number of instances, was somewhat curved, being described as either kidney-shaped or sausage-shaped.

In only one instance was the correct diagnosis made before operation, Krabbel's second case. In thirteen of the twenty-two cases a mass in the upper part of the right side of the abdomen was felt, and in eight

cases it was stated that a mass could not be made out. In eight instances the preoperative diagnosis was acute cholecystitis, in three cases ileus was suspected, and in one patient a twisted ovarian cyst was believed to be present. In several the condition was thought to be acute appendicitis. In considering this mistaken diagnosis, it should be remembered that many old and thin women have a marked downward displacement of the liver that brings the gallbladder near the region of the appendix.

In twelve cases of this series it was specifically stated that jaundice was not present, and its presence was not noted in any instance. This is not surprising, as the torsion was in the cystic duct and little evidence of infection was present, except in Wendel's case.

In two reports the age of the patients was not given; in the other cases all the patients except five were over 60 years old. These five were 23, 34, 42, 54, and 54, respectively; nine were 70 or over.

Twenty-one of the twenty-two patients were operated on, Kubig's case being the only exception. Cholecystectomy was performed in each instance, and in each instance the subhepatic area was drained. Removal of the gallbladder was easily performed, the only operative difficulty being in Hansen's case, in which there was an hour-glass gallbladder. Of these twenty-one patients operated on, seventeen recovered.

Stones were found in the gallbladder in seven cases, and in none was there an impacted stone in the cystic duct with hydrops. Stones were not reported in either the hepatic or the common ducts. It appears that biliary calculi do not play a part in torsion, and that their presence is incidental.

Fever was not a constant symptom, and there was great variation in the pulse rate in the different patients. Symptoms of shock were not commonly present. In one case there was severe shock, which continued until death.

In the majority of the cases the onset was sudden, and in many instances there had not been any upper abdominal discomfort previously. The outstanding symptom was pain, often followed by vomiting. Altogether, the clinical picture is a definite one, but it is so rarely encountered that the likelihood of this condition being present is usually not considered.

CARDIAC OUTPUT IN THE DOG DURING ETHER ANESTHESIA

III. THE EFFECT OF THERAPEUTIC AMOUNTS OF DIGITALIS ON CARDIAC OUTPUT OF THE ANESTHETIZED DOG *

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In previous communications,[1] evidence has been presented to show that when ether is administered to the correct degree for surgical anesthesia, it causes an increase in the cardiac output, and that this increase is not so great after alkali has been injected as before. It was concluded from this work that a large part of the increase in the cardiac output of the heart during ether anesthesia was caused by an increase in the hydrogen ion concentration of the blood. However, the less marked but definite increase in the cardiac output of the anesthetized dog following the injection of alkali suggests that part of the increase must be ascribed to a direct stimulating action of ether on the heart.

Harrison and Leonard [2] have shown recently that digitalis decreases the cardiac output of dogs, and their evidence indicates that this is accomplished primarily through the production of an increased tonicity of the heart. The effects on the contractility were believed to be secondary to changes in the tonus. It is generally conceded that digitalis exerts its beneficial effect by action on the heart itself, regardless of whether this is accomplished through changes in the cardiac output or through a restoration of balance between the ventricles. Eppinger, von Papp and Schwarz [3] reported a decrease in the cardiac output in one patient following the administration of digitalis. Studies by indirect methods by many observers have indicated that digitalis increases the output of the heart in animals that have had various operative procedures performed; but concerning the effect on the normal dog, the work of Harrison and Leonard seems conclusive.

The purpose of the present study was to determine the effect of ether anesthesia on the cardiac output of the digitalized dog. This was undertaken with two objects in view: (*1*) to attempt to gain further information as to what direct stimulating action ether has on the heart

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1. Blalock, A.: The Effect of Ether Anesthesia on the Cardiac Output of the Dog, Arch. Surg. **14**:732 (March) 1927. The Effect of the Injection of Alkali on the Cardiac Output of the Anesthetized Dog, Ibid. **14**:921 (April) 1927.

2. Harrison, T. R., and Leonard, B. W.: The Effect of Digitalis on the Cardiac Output of Dogs, J. Clin. Investigation **3**:1 (Oct.) 1926.

3. Eppinger, Hans; von Papp, L., and Schwarz, H.: Asthma Cardiale, Berlin, Julius Springer, 1924, p. 191.

itself, since it has been shown that digitalis decreases the output of the heart and ether increases it somewhat even in the presence of an alkaline p_H; (*2*) to determine whether there is experimental justification for the use of digitalis previous to operations under ether anesthesia.

METHOD

Only the essential features of the method which was used will be described, since a detailed description has been given in the report on the effect of ether anesthesia on the cardiac output.

Thirty-six determinations of the cardiac output were made in six dogs. Two of the animals were trained and remained quiet without a sedative. The remaining four dogs were given morphine, 0.04 to 0.06 Gm., about an hour before the experiment was begun. Similar results were obtained with and without the use of morphine.

In five of the six experiments, control studies were performed before digitalis was given in order to determine the effect of ether anesthesia on the cardiac output. In the remaining experiments the effect of a digitalis preparation and ether anesthesia on the cardiac output was first determined, and the control figure for ether alone was obtained four days later. The time interval separating the intramuscular injection of digitalis and the determinations under ether anesthesia varied from six to twenty-two hours. Harrison and Leonard[3] found that the maximum reduction in cardiac output occurred six hours after administering a "full therapeutic" dose of digitalis preparation, and that it lasted for about forty-eight hours. The amount of the drug used was approximately the same as that employed in their experiments, i. e., fifteen cubic centimeters of digitalis preparation per kilogram of body weight. Pardee's[4] observations indicate that this amount represents the full therapeutic dose.

The animal was placed on a table in the dorsal position. The oxygen consumption was determined by use of a Benedict spirometer with the graphic recording device. Arterial blood was obtained by puncture of the left ventricle or femoral artery; venous blood was obtained by puncture of the right ventricle. The oxygen and carbon dioxide contents of the blood were determined. The circulatory minute volume was calculated from the Fick formula:

$$\frac{\text{Cc. } O_2 \text{ consumed per minute}}{\text{Amount } O_2 \text{ taken up in lungs by 1 cc. of blood}} = \text{Number of cc. of blood flowing through the lungs per minute}$$

After the control determinations had been made, ether was administered through an open cone. Variations in the depth of anesthesia are listed in the protocols, but usually the lid reflexes were abolished at the time that the specimens of blood were drawn. The method of Austin[5] was employed for determining the carbon dioxide content in the presence of ether.

RESULTS

Both the trained animals and those that had undergone morphinization remained quiet throughout the experiments, and the results were essentially the same with and without the use of morphine.

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4. Pardee, H. E. B.: Hypodermia Digitalis Preparations, J. A. M. A. **85**:1359, (Oct. 31) 1925.

5. Austin, J. H.: Estimation of Carbon Dioxide in Serum in Presence of Ether by Van Slyke Method, J. Biol. Chem. **61**:345 (Sept.) 1924.

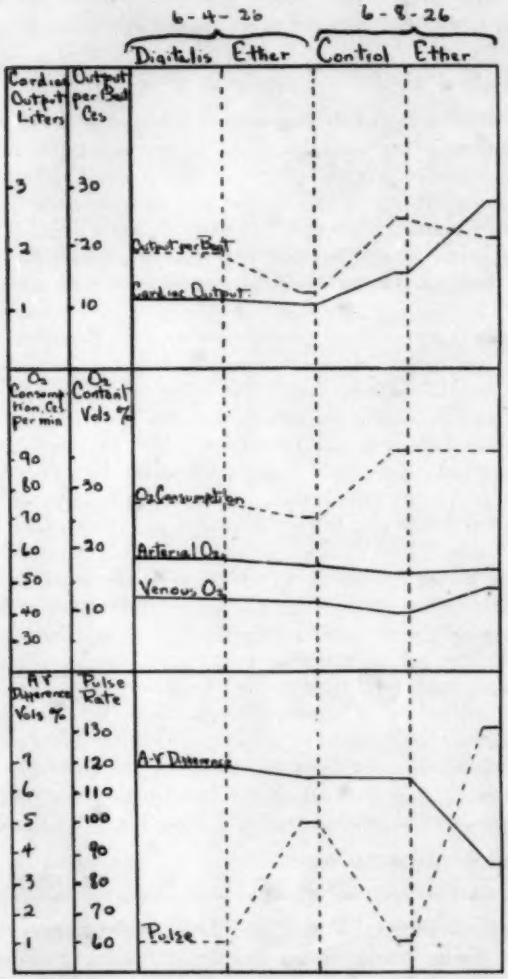

Chart 1.—The cardiac output of the digitalized dog remained about the same during ether anesthesia. Four days later, the output increased markedly during ether anesthesia. No digitalis was given during this interval. The oxygen consumption was higher after the effects of digitalis had disappeared. The coefficient of utilization changed little during an ether anesthetic seven and one-half hours after digitalis had been given, but decreased greatly when the dog was anesthetized four days later.

The drawings are purely schematic; time relations are neglected.

The changes which were encountered during the control anesthetic period will not be described in detail. The most striking of these were the increased output of the heart and the decrease in the coefficient of utilization (charts 1 and 2, tables 1 to 6).

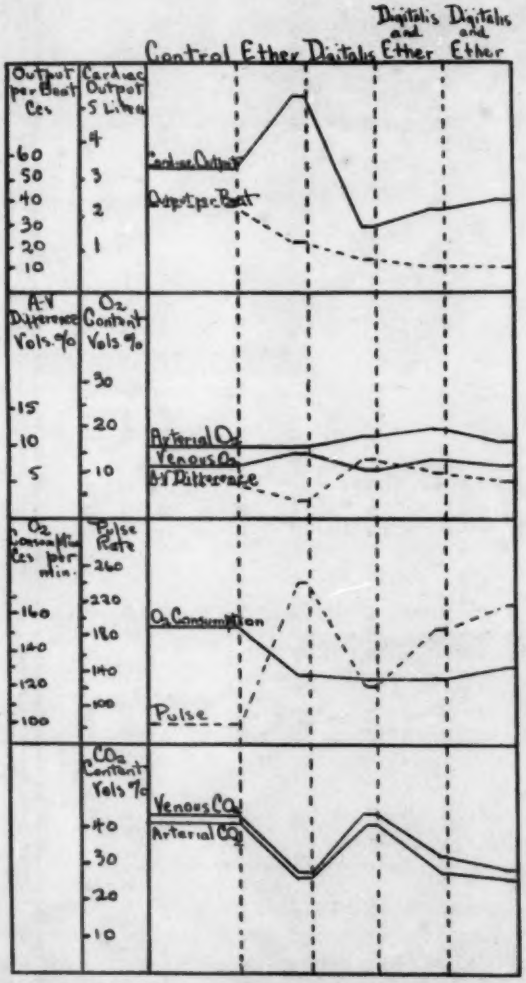

Chart 2.—This figure illustrates the reduction in cardiac output following the administration of digitalis and the slight increase when ether is given, whereas previously ether had caused a great increase in the output. The pulse rate rose during each anesthetic period, the cardiac output per beat decreased, and the carbon dioxide content of the blood fell.

This drawing is purely schematic; time relations are ignored.

The effects of digitalis on the cardiac output of a dog that has not been anesthetized are thought not to be absolute in these experiments, because in most instances the animal had been given ether a few hours

TABLE 1.—*The Effect on the Cardiac Output of Ether and of Digitalis and Ether*

Date	Weight, Kg.	Temperature, Fahrenheit	Pulse Rate per Minute	Arterial Oxygen, per Cent by Volume	Venous Oxygen, per Cent by Volume	Arteriovenous Difference, per Cent by Volume	Oxygen Capacity, per Cent by Volume	Per Cent Saturation	p_H	Oxygen Consumption, Cc. per Minute	Cardiac Output, Cc. per Minute	Output per Kg. per Minute, Cc.	Output per Beat, Cc.
5/12/26: Control study, ¾ grain morphine	8.6	99	70	15.36	8.88	6.48	17.1	90	7.35	73.95	1,141	132.7	16.3
5/12/26: After 7 minutes ether	8.6	99	150	14.04	11.04	3	17.22	82	7.29	59.04	1,968	228.9	13.1
5/12/26: After additional ether	8.6	99	150	14.40	11.40	3	.....	..	7.30	58.4	1,947	226.4	13.0
5/12/26: 2 hours after stopping ether	8.6	98.5	76	15.24	8.66	6.58	.....	..	7.36	75.64	1,148	133.4	15.1
5/15/26, 2 p. m.: Control study, ¾ grain morphine	8.2	101	120	13.8	6.97	6.83	14.6	94.5	....	62.52	915	111.5	7.6
5/15/26, 3:15 p. m.: After 6 minutes anesthesia.....	8.2	101	180	11.51	8.36	3.15	17.0	68	....	57.2	1,810	221	10.1
5/15/26, 10:30 p. m.: 6½ hours after 4.2 cc. of a digitalis preparation	8.2	100.6	210	13.2	4.85	8.35	15.02	89	....	88.15	1,056	128.7	5.02
5/15/26, 11:55 p. m.: After 7 minutes anesthesia	8.2	100.6	225	11.26	5.70	5.56	13.6	83	....	60.88	1,095	133.5	4.9
5/16/26, 11:30 a. m.: 20 hours after digitalis preparation	8.1	101	210	11.22	4.20	7.02	11.34	99	....	82.0	1,168	144	5.5
5/16/23, 2 p. m.: After 7 minutes anesthesia	8.1	99.6	220	10.08	5.65	4.43	12.48	87	....	61.0	1,377	170	6.3
5/16/26, 5:15 p. m.: After 75 minutes anesthesia....	8.1	97.6	225	9.08	3.60	6.20	11.4	86	....	66.4	1,071	132	4.7

Protocol: 5/12/26: A control study was performed after three fourths grain (0.04 Gm.) of morphine was given. The dog was then given ether anesthesia through a mask for seven minutes until the eye reflexes were barely abolished, and determination of the output were made. The ether was discontinued for a short period; ether was again administered for seven minutes, and the output was determined. The eye reflexes were abolished. Two hours after the anesthetic was stopped, a second control study was made.

5/15/26: At 2:00 p. m., a control study was made after three fourths grain (0.04 Gm.) of morphine was given. Ether anesthesia was begun at 3:15 p. m., and determinations were made six minutes later, the eye reflexes being abolished at the time the specimens of blood were obtained. At 4:00 p. m., the animal was given 4.2 cc. of a digitalis preparation. At 10:30 p. m., six and a half hours following the giving of the drug, one half grain of morphine (0.03 Gm.) was given, and control studies were made. At 11:55 p. m., anesthesia was given for seven minutes, eye reflexes were abolished, and the determinations were made.

5/16/26: At 11:30 a. m., twenty hours following the giving of a digitalis preparation, one half grain of morphine (0.03 Gm.) was given, and control studies were made. At 2:00 p. m., anesthesia was given for six minutes, eye reflexes were abolished, and control determinations were made. At 5:15 p. m., determinations were made after anesthesia for one hour and fifteen minutes, and the eye reflexes were abolished. The pulse was regular and rapid.

previously, and this may have altered the result. The cardiac output was the same as in the original control period in two instances, slightly elevated in one and decreased in three instances. In general, the results are confirmatory of the experiments of Harrison and Leonard.

The following is a comparison of the cardiac output during ether anesthesia in the digitalized dog with the figures obtained under other conditions:

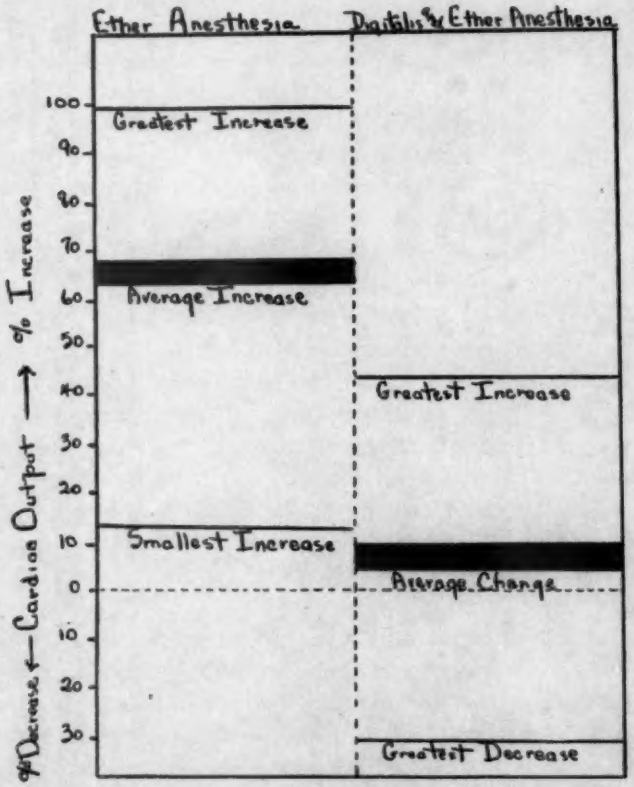

Chart 3.—The percentage change in cardiac output during the first anesthesia period was determined by comparing the figure for cardiac output per minute with the one obtained during the control period, and the cardiac output during the second anesthesia period was compared with that following the administration of digitalis. The average change was computed from the figures in all of the experiments. The smallest change during ether anesthesia without digitalis was greater than the average change during ether anesthesia in the digitalized dog.

1. The cardiac output remained essentially the same as in the original control period in six determinations, rose slightly in one, fell slightly in three and decreased definitely in two instances. The average cardiac output per kilogram of body weight was only 4 cc. different from that of the original control period.

TABLE 2.—*The Effect on the Cardiac Output of Ether and of Digitalis and Ether*

Date	Weight, Kg.	Temperature, Fahrenheit	Pulse Rate per Minute	Arterial Oxygen, per Cent by Volume	Venous Oxygen, per Cent by Volume	Arteriovenous Difference, per Cent by Volume	Arterial Carbon Dioxide, per Cent by Volume	Venous Carbon Dioxide, per Cent by Volume	Oxygen Capacity, per Cent by Volume	Oxygen Consumption, Cc. per Minute	Cardiac Output, Cc. per Minute	Output per Kg. per Minute, Cc.	Output per Beat, Cc.
5/25/26, 8:15 a. m.: Control study	10.5	102	90	19.16	13.55	5.61	34.46	37.72	.....	103.3	1,841	175	20
5/25/26, 4 p. m.: After ether for 17 minutes	10.5	102	215	20.60	16.69	3.91	24.44	28.08	.....	99.6	2,547	242	12
5/26/26, 4:00 p. m.: Control study	10.5	101.6	108	19.11	12.62	6.49	.....	.....	.....	118.1	1,820	173	17
5/26/26, 10:30 p. m.: 6 hours after 5.5 cc. digitalis preparation	10.5	104.4	95	17.35	11.88	5.47	34.30	38.45	.....	125.5	2,285	218	24
5/26/26, 11:55 p. m.: Ether 35 minutes	10.5	104	200	18.06	10.45	7.61	26.26	30.83	18.23	118.1	1,552	148	8
5/27/26, 2:30 p. m.: 22 hours after digitalis	10.1	104	125	16.4	9.31	7.09	35.91	40.20	99%	147.6	2,082	206	17
5/27/26, 4:15 p. m.: 15 minutes ether	10.1	102	220	18.16	12.97	5.19	31.49	34.17	18.26 99%	103.3	1,990	197	9

Protocol: 5/25/26: A healthy dog was used. At 8:15 a. m. control determinations were made without morphine. An attempt was then made to anesthetize the dog by giving ether and cotton seed oil per rectum. This was unsuccessful. At 3:45 p. m., the dog was given ether by mask for seventeen minutes before specimens of blood were taken; eye reflexes were barely obtainable. 5/26: At 4:00 p. m., a control study was made without morphine; the animal was given 5.5 cc. of a digitalis preparation after determinations. At 10:30 p. m., six hours following the giving of a digitalis preparation, determinations were made. At 11:55 p. m., determinations were made after thirty-five minutes of ether anesthesia; eye reflexes were present. The animal began to move about four minutes after the ether was stopped. 5/27: Twenty-two hours following the giving of digitalis, at 2:30 p. m., determinations were made without ether. At 4:15 p. m., determinations were made after ether anesthesia for fifteen minutes; the eye reflexes were not abolished.

2. The cardiac output was less in every instance than that during the original anesthetic period. The average figure for the cardiac output per kilogram of body weight was 243 cc. during the first anesthetic period, while that during ether anesthesia following the administration of a digitalis preparation was only 147 cc. The largest figure obtained during the latter period was smaller than the average figure during the first anesthetic period. These alterations are represented in terms of percentage of change in chart 3.

3. The changes which ether anesthesia produced on the cardiac output of the digitalized dog are almost negligible. This is demonstrated by the fact that the average cardiac output per kilogram of body weight was 148 cc. before the administration of ether, and 147 cc. during the period that the animal was under the influence of an anesthetic.

Changes in pulse rate, oxygen consumption and carbon dioxide content were in general similar to those during the anesthetic period without digitalis, but the coefficient of utilization was greater in each instance.

COMMENT

In view of the fact that experiments were performed on trained dogs as well as on those anesthetized with morphine, and that the results were similar in each instance, it seems justifiable to state that digitalis prevents an increase in the cardiac output of the anesthetized normal dog. It is known that most drugs have the same type of effect on man as on the dog. In order to discuss the possible general significance of this experimental work, it is necessary to assume that such a relation exists because of the difficulties and dangers in determining the cardiac output of man.

Recent work supports the view that digitalis exerts its beneficial effect by maintaining a balance between the two ventricles. Cushny[6] stated that digitalis exerts little influence on the blood pressure. It has been found in studying the effect of ether anesthesia on the cardiac output that all of the increase could not be due to the effect of changes in the reaction of the blood, and that there is a direct stimulating action by ether on the heart was assumed. Practically no increase in cardiac output resulted when ether was administered to dogs that had received digitalis. If it is granted that digitalis decreases the cardiac output by direct action on the heart, this is further evidence that part of the elevation in the cardiac output during ether anesthesia is caused by a specific stimulating effect of the anesthetic on the heart. Determinations of the hydrogen ion concentration were not made in these experiments, but it cannot be assumed that digitalis prevents the usual increase, especially since the carbon dioxide content behaved in the usual manner. It seems

6. Cushny, A. R.: The Action and Uses in Medicine of Digitalis and Its Allies, London, Longmans, Green & Co., 1925, p. 117.

TABLE 3.—*The Effect on the Cardiac Output of Ether and of Digitalis and Ether*

Date	Weight, Kg.	Temperature, Fahrenheit	Pulse Rate per Minute	Arterial Oxygen, per Cent by Volume	Venous Oxygen, per Cent by Volume	Arteriovenous Difference, per Cent by Volume	Arterial Carbon Dioxide, per Cent by Volume	Venous Carbon Dioxide, per Cent by Volume	Oxygen Capacity, per Cent by Volume	Oxygen Consumption, Cc. per Minute	Cardiac Output, Cc. per Minute	Output per Kg. per Minute, Cc.	Output per Beat, Cc.
6/4/26, 9:30 p. m.: 7½ hours after digitalis	10.2	98.5	60	18.48	11.76	6.72	38.19	39.97	.....	75.64	1,125	110.3	18.7
6/4/26, 10:30 p. m.: After 25 minutes ether	10.2	97.2	100	17.88	11.58	6.30	37.10	42.15	19.74 91%	70.11	1,113	109.1	11.1
6/8/26, 2:30 p. m.: 1 grain morphine; 3:40 p. m., control	9.8	99.5	60	15.98	9.66	6.32	38.78	41.36	.....	92.2	1,459	149	24.3
6/8/26, 4:50 p. m.: After 30 minutes ether	9.8	97.6	122	16.58	13.0	3.58	39.05	40.82	17.47 95%	92.2	2,771	282.8	21

Protocol: 6/4/26: At 2:00 p. m., a healthy dog was given 6.5 cc. of a digitalis preparation. At 8:30 p. m., it received 1 grain (0.06 Gm.) of morphine. At 9:30 p. m., the dog was perfectly quiet and the cardiac output was determined. At 10:30 p. m., determinations were made after administration of ether for twenty-five minutes. The eye reflexes were not abolished. 6/8/26: At 2:30 p. m., the animal received 1 grain (0.06 Gm.) of morphine. At 3:40 p. m., the cardiac output was determined. At 4:50 p. m., the output was determined after ether had been administered for thirty minutes. The eye reflexes were not abolished.

TABLE 4.—*The Effect on the Cardiac Output of Ether and of Digitalis and Ether*

Date	Weight, Kg.	Temperature, Fahrenheit	Pulse Rate per Minute	Arterial Oxygen, per Cent by Volume	Venous Oxygen, per Cent by Volume	Arteriovenous Difference, per Cent by Volume	Arterial Carbon Dioxide, per Cent by Volume	Venous Carbon Dioxide, per Cent by Volume	Oxygen Capacity, per Cent by Volume	Oxygen Consumption, Cc. per Minute	Cardiac Output, Cc. per Minute	Output per Kg. per Minute, Cc.	Output per Beat, Cc.
6/12/26, 2:30 p. m.: Control study; no morphine	14	103	84	15.8	10.96	4.84	41.59	43.99	.....	151.29	3,126	223.3	37.2
6/12/26, 5:00 p. m.: After 2 hours ether	14	102.6	240	15.8	13.36	2.44	23.33	24.66	.....	125.46	5,142	367	21.4
6/13/26, 11:30 a. m.: 12½ hours after digitalis	14	102.4	120	17.69	10.41	7.28	38.62	43.76	.....	121.77	1,673	119.2	14
6/13/26, 2:35 p. m.: After 35 minutes ether	14	103	180	18.04	11.94	6.10	26.10	30.18	18.10 90.7%	121.77	1,996	143	11
6/13/26, 4:00 p. m.: After 2 hours ether	14	102.6	210	15.69	10.3	5.39	24.12	27.54	.....	129.15	2,396	171	11.4

Protocol: 6/12/26: At 2:30 p. m., control studies were made on a trained dog without the use of morphine. At 3:00 p. m., ether anesthesia was begun. At 5:00 p. m., determinations were made during ether anesthesia, eye reflexes being barely obtainable. 6/13: At 11:30 a. m., control determinations were made twelve and a half hours following the injection of 7 cc. of a digitalis preparation. At 2:00 p. m., ether anesthesia was begun. At 2:35 p. m., determinations were made during ether anesthesia, eye reflexes were barely obtainable. At 4:00 p. m., determinations were made, two hours following the beginning of the ether anesthesia. The eye reflexes were barely obtainable.

probable that the depressing effect of digitalis on the output of the heart counteracts part of the stimulating influence which ether produces. The heart of the dog that has received digitalis is much less subject to fluctuations in output, as is demonstrated by the small variation in output when ether is administered. This is shown in chart 4. It is to be noted not only that the average of all the figures for cardiac output per kilogram of body weight are practically identical, but also that the highest and lowest figures obtained in all of the experiments fall within a rather small range.

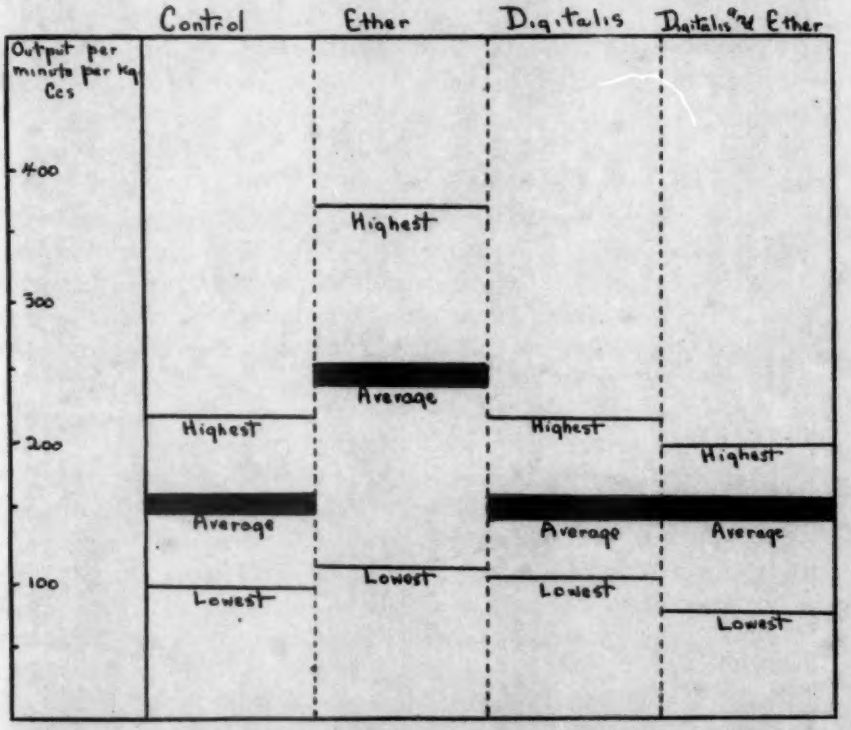

Chart 4.—This is a composite chart of all experiments. The highest and lowest values per kilogram of body weight during the four phases of the experiments are charted on the thin lines. The average output is not the mean of the highest and lowest values, but is the average of all the figures. It is thought that the cardiac output during the control period following the giving of digitalis would have been lower if ether had not been administered previously. There is a striking rise in cardiac output during ether anesthesia without digitalis.

Harrison and Leonard have found that the elevation in the cardiac output which is present in dogs with hyperthyroidism and anemia can be decreased by the administration of digitalis. They express the belief that this drug would probably be valuable in preventing cardiac failure from overstrain in these conditions. Apparently, digitalis establishes a

TABLE 5.—*The Effect on the Cardiac Output of Ether and of Digitalis and Ether*

Date	Weight, Kg.	Temperature, Fahrenheit	Pulse Rate per Minute	Arterial Oxygen, per Cent by Volume	Venous Oxygen, per Cent by Volume	Arteriovenous Difference, per Cent by Volume	Arterial Carbon Dioxide, per Cent by Volume	Venous Carbon Dioxide, per Cent by Volume	Oxygen Capacity, per Cent by Volume	Oxygen Consumption, Cc. per Minute	Cardiac Output, Cc. per Minute	Output per Kg. per Minute, Cc.	Output per Beat, Cc.
6/30/26, 3:20 p. m.: Control study	8.5	99.8	100	18.08	10.11	7.97	36.72	42.66	.....	67.65	849	100	8.5
6/30/26, 4:35 p. m.: After 50 minutes anesthetic	8.5	99	140	18.91	11.41	7.50	34.43	38.88	19.0	71.75	957	113	7
7/1/26, 5:10 p. m.: 24 hours after digitalis preparation	8.5	99	72	18.79	12.13	6.66	40.10	42.80	99%	61.5	923	109	13
7/1/26, 6:05 p. m.: After 15 minutes anesthetic	8.5	210	96.6	14.87	6.42	8.45	39.93	42.23	.....	61.5	728	86	8

Protocol: 6/30/26: At 2:00 p. m., three fourths grain (0.04 Gm.) of morphine was given. At 3:20 p. m., control study was made. At 3:45 p. m., ether was begun. At 4:30 p. m., eye reflexes were abolished, and specimens of blood were drawn. At 5:00 p. m., the dog was given 5 cc. of a digitalis preparation. 6/31/26: At 4:00 p. m., three fourths grain (0.04 Gm.) of morphine was given. At 5:10 p. m., twenty-four hours after a digitalis preparation was given, control studies were made. At 5:30 p. m., ether anesthesia was begun. At 6:05 p. m., eye reflexes were abolished, and specimens of blood were drawn.

TABLE 6.—*The Effect on the Cardiac Output of Ether and of Digitalis and Ether*

Date	Weight, Kg.	Temperature, Fahrenheit	Pulse Rate per Minute	Arterial Oxygen, per Cent by Volume	Venous Oxygen, per Cent by Volume	Arteriovenous Difference, per Cent by Volume	Arterial Carbon Dioxide, per Cent by Volume	Venous Carbon Dioxide, per Cent by Volume	Oxygen Consumption, Cc. per Minute	Cardiac Output, Cc. per Minute	Output per Kg. per Minute, Cc.	Output per Beat, Cc.
7/19/26, 3:00 p. m.: Control study, morphine ¾ grains	13.5	102	60	8.46	3.90	4.56	39.16	42.64	97.2	2,132	158	35.5
7/19/26, 3:50 p. m.: After ether for 35 minutes	13.5	101.6	120	8.72	6.27	2.45	39.02	42.24	86.4	3,527	263	29.3
7/23/26, 2:15 p. m.: 14 hours after 6.5 cc. digitalis preparation	13	100.8	50	9.31	4.71	4.60	.....	.....	90.0	1,937	151	39
7/23/26, 3:50 p. m.: After 35 minutes ether	13	101	120	8.72	5.30	3.42	36.12	37.90	78.0	2,281	175	19
7/23/26, 4:25 p. m.: After 70 minutes ether	13	101	135	8.60	4.71	3.89	.....	.....	75.6	1,943	149	14

Protocol: 7/19/26: At 2:00 p. m., three fourths grain (0.04 Gm.) of morphine was given. At 3:00 p. m., the animal was quiet, and control determinations were made. The dog looked perfectly well but was anemic, the cause of which was undetermined. At 3:15 p. m., ether anesthesia was begun. Specimens of blood were drawn at 3:50 p. m. after thirty-five minutes of anesthesia, the eye reflexes were abolished at the time. 7/23: At 12:15 p. m., a digitalis preparation, 6.5 cc., was given. At 12:30 p. m., three fourths grain (0.04 Gm.) of morphine was administered. At 2:15 p. m., control determinations were made. At 3:15 p. m., ether anesthesia was begun. At 3:50 p. m., the eye reflexes were abolished and specimens of blood were drawn for determinations. At 4:25 p. m., the eye reflexes were abolished, and determinations were made after anesthesia for seventy minutes.

balance at a lower level and probably protects the weaker ventricle from overstrain by the stronger one. For the same reason it seems that the preoperative administration of digitalis should be of definite therapeutic value under the following circumstances:

1. In patients suffering from conditions known to be associated with an increased cardiac output. Such conditions are hyperthyroidism, severe anemia, anoxemia [7] and probably other disorders still to be investigated.

2. In patients of all ages with hypertension or organic heart disease.

3. In elderly and debilitated patients.

Marvin, Pastor and Carmichael [8] have concluded from electrocardiographic and blood pressure studies during surgical operations and convalescence that routine employment of digitalis in patients with normal hearts is not necessary.

Since it is well known that shock results in a decrease in the cardiac output, the administration of digitalis should not be delayed until after this condition is produced. The rôle of digitalis should be to prevent acute circulatory failure rather than to combat it; hence, if digitalis is given, it should be administered previous to the anesthetic. It is felt that when given under such conditions, digitalis may protect the heart from overstrain by increasing its tonus and diminshing the contractility. Digitalis has been used rather generally in the past in the preparation of patients with heart disease for operation, but usually with a different aim. For example, Richardson [9] stated that digitalis should be used to alleviate the period of depression following operation. Rather than this, it seems that digitalis should serve to prevent strain on the heart at the time of operation and by this means to prevent the depression which might follow. In a previous paper,[10] it was shown that the administration of alkali partially prevented the increase in cardiac output. Digitalis is preferable to alkali for two reasons: 1. The desired dosage of digitalis is fairly well known, whereas the amount of alkali which is needed is dependent on the acidosis, which cannot be estimated with accuracy previous to the operation. 2. Digitalis exerts its influence essentially on

7. Harrison, T. R., and Blalock, A.: Studies on the Effects of Hyperthyroidism, Anemia and Anoxemia on the Cardiac Output, unpublished observations.

8. Marvin, H. M.; Pastor, R. B., and Carmichael, Mabel: Electrocardiogram and Blood Pressure During Surgical Operation and Convalescence; Effect of Routine Preoperative Digitalization, Arch. Int. Med. **35**:782 (June) 1925.

9. Richardson, F. L.: Heart Serums in Anesthesia, Am. J. Surg. anesthesia suppl. **33**:109 (Oct.) 1919.

10. Blalock, A. (footnote 1, second reference).

the heart, whereas it is possible that the administration of alkali, though tending to prevent cardiac strain, may lead to untoward effects elsewhere in the body.

In this connection it is emphasized that the full therapeutic dose of digitalis was given in these experiments, and if a similar effect is to be expected in patients, doses of similar size are indicated. Such a dose is approximately 1.5 Gm. of a powdered leaf of average strength, or fifteen cubic centimeters of a digitalis preparation per hundred pounds of body weight. In order to increase the margin of safety, it seems wiser to administer two thirds of this amount in divided doses the day previous to operation.

SUMMARY AND CONCLUSIONS

The effect of ether on the cardiac output of six digitalized dogs was determined. Four of the animals received morphine previous to the studies, while two of the animals were trained and did not require morphine. Whereas the average elevation in the cardiac output during ether anesthesia previous to the administration of digitalis was 64 per cent, that in the digitalized dog was only 7 per cent. Further evidence is presented to show that part of the increase in the cardiac output during ether anesthesia is caused by a direct stimulating action of ether on the heart.

These observations offer a rational experimental basis for the belief that the preoperative administration of digitalis may be of considerable value in the preparation of certain types of patients for operation under general anesthesia. They also suggest that this drug should not be used during shock following operations.

STOMACH MUCOSA IN ULCER AND IN CARCINOMA

HISTOLOGIC STUDIES *

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Former histologic studies of the stomach mucosa have been based chiefly on the examination of postmortem material. Later investigators have introduced fixing fluids into the stomach at various periods after death in order to prevent self-digestion of the stomach mucosa. There are on record the results of examinations of stomachs immediately after capital punishment (Kaufmann [1]). Reports of the results of examinations of stomachs resected for cancer (Matti [2]), or of examinations of parts of stomachs removed during gastro-enterostomy (Heyrovsky [3]) have also appeared at various times. Only during the last few years have surgeons treated ulcers of the stomach and duodenum by gastrectomy. Stoerk,[4] Konjetzny,[5] Moszkowicz [6] and Orator [7] have availed themselves of the surgical material and have made comprehensive reports of their observations.

The work reported in this paper was started at the University of Vienna, department of histology and bacteriology, under the direction of the late Professor Stoerk. The material examined was obtained chiefly from patients operated on by Professor Finsterer. Some of the last series of the stomachs examined were those of patients on whom I had performed operations.

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* From the Pathology and Physiology Departments of the University of Oregon Medical School.

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5. Konjetzny, G. Ernest: Chronische Gastritis und Duodenitis als Ursache des Magenduodenol geschwürs, Beitr. z. path. Anat. u. z. allg. Path. **71**:595, 1923.

6. Moszkowicz, Ludwig: Zur Histologie des ulcusbereiten Magens Arch. f. klin. Chir. **122**:444, 1922.

7. Orator, Victor: Ueber den Ulcus und Carcinommagen, Wien. klin. Wchnschr. **16**:425 (April) 1925. Beiträge zur Magenpathologie. IV. Arch. f. klin. Chir. **134**:663, 1925.

In all, twenty stomachs were examined—twelve were resected for ulcer of the duodenum and ulcer of the stomach, and eight for cancer. Immediately after resection most of the stomachs were introduced into a solution containing one part of solution of formaldehyde and two parts of 80 per cent alcohol. The stomachs resected were opened along the greater curvature. Sections were taken along the whole circumference, in the region of the pylorus, midway between the pylorus and at the proximal edge.

In addition to this systematic study, parts of the tissue from the mucosa of other stomachs were examined, including eight resected gastric ulcers and six resected duodenal ulcers. The sections were stained with hematoxylin-eosin. Special stains were used occasionally to demonstrate points of interest.

I shall give a clinical and pathologic report of one case and present my histologic observations of the other material according to the following classification: (1) normal stomach mucosa, (2) état mammelonné, (3) atypical characteristics of the stomach mucosa, including intestinal islands and erosions, (4) the nature of the inflammation of the stomach mucosa and (5) comment.

Anatomists are somewhat at variance as to the nomenclature of the parts of the stomach. In order to discuss the histologic observations, I shall divide the stomach into the large proximal part—which includes the part long known as fundus and corpus—the pars digestoria, and the distal part, which is smaller, contains the pyloric glands, and is known as the antrum or pars egestoria. The terms fundus, corpus and pars digestoria will be used interchangeably to indicate the proximal part. The pars egestoria and the antrum will also be used interchangeably to indicate the distal part of the stomach.

REPORT OF A CASE

History.—N. G., white, aged 28, a miner, was admitted to St. Vincent's Hospital on Sept. 15, 1923. He was well until six months before, when he began to vomit. He became much worse a few weeks before coming to the hospital, vomiting frequently, immediately after eating and sometimes several hours later. He never vomited blood or coffee-ground material and never noticed black stools. He never experienced pain in his adbomen. He lost about 40 pounds (18 Kg.) during the last few months before entering the hospital, and was quite weak. His appendix had been removed in 1921.

Examination.—He was emaciated. The abdomen was fat; there was a tympanitic note and a splashing sound in the region of the epigastrium. There was no rigidity or tenderness in the abdominal wall. The scar of the appendectomy was found in the right lower quadrant.

Gastric analysis showed a high acidity. The hemoglobin content was 72 per cent; the white blood count was 8,350. There was a trace of albumin in the urine.

Roentgen-Ray Examination (Dr. Diemer).—Although the patient said that he had not eaten for fifteen hours, a quantity of material was observed in the stomach. Roentgenograms taken after the ingestion of barium meal revealed that the stomach was increased in size and capacity and was boat-shaped on account of the dilatation. A pathologic gastric condition was not otherwise demonstrated.

The first portion of the duodenum did not fill. Only a small stream of barium was observed in the duodenum. Roentgen-ray examination indicated obstruction as a result of chronic fibrous ulcer of the duodenum. Adhesions of the stomach were also considered.

Operation and Course.—I operated on this patient on September 17, after two days of frequent lavage of the stomach. Anterior splanchnic anesthesia was used. The pylorus was thick, indurated with numerous thick adhesions about the antrum and duodenum. When the distal part of the stomach was resected, two ulcers were found in the first part of the duodenum. A modification of Billroth's second operation (Hofmeister-Finsterer), was performed. The patient felt well for a few days, but later developed symptoms of bronchopneumonia; these, however, soon cleared up. The patient did not vomit. He gained 10 pounds (4.5 Kg.) during his stay, and left the hospital on October 24.

I saw the patient two months later. He had gained 40 pounds and had no symptoms of any kind. He was working as a laborer and did not adhere to any particular diet. A roentgen-ray examination made by Dr. Diemer at that time revealed that the esophagus was normal; in other words, it did not dilatate at its distal end as a compensatory measure for the lessened capacity of the stomach. Apparently, at least one half of the stomach had been removed. The remaining portion presented perfectly smooth borders. A pathologic gastric condition was not indicated by any defect in filling. It was observed that the cardiac part of the stomach developed a compensatory peristaltic action. Emptying began immediately and continued in a normal moderate manner. The stomach was freely movable, and a pathologic condition of the duodenum was not evident.

The chemical examination at this time showed that the stomach did not contain free acid.

Pathologic Report.—The gross examination of the resected specimen revealed a deep indurated ulcer on the anterior wall of the first part of the duodenum with thickening of the serosa about it. There was a scar with evidence of previous ulceration near the large ulcer.

Microscopic examination of the mucosa in the region of the antrum, 1 cm. proximal to the pyloric ring, demonstrated a fan-shaped arrangement of the groups of glands, the foveolae being long and straight. Only two or three alveoli opened into the tubule; the cells lining the foveolae were light-staining; the interstitial tissue betwen the foveolae and the tubules was rather scant. The muscularis mucosa reached high between the groups of glands; the lymph follicles were triangular and not numerous. Deep valleys reaching the muscularis mucosa appeared where the epithelium was desquamated and where there was infiltration of round cells.

Dark-staining epithelium suggested previous destruction of glands and a process of regeneration; otherwise the glands appeared normal. On the inferior surface, about 1 cm. from the pyloric ring, some glands appeared normal, and corresponded to the description given. There were, however, groups of glands with tall foveolae. Branched alveoli were found in connection with each tubule, the cells lining the alveoli were cuboidal, the nuclei were small, found at the

base; the lymph follicles were irregular and increased in size; there were also groups of round cells. The interstitial tissue between the alveoli was increased; dark-stained epithelium with goblet cells were found near these groups of glands. Some of the round cells penetrated into the muscularis mucosa (fig. 1).

Sections made through the same area demonstrated an ulcer which was not found at the examination of the gross specimen with the naked eye. The ulcer was about 2 mm. in diameter. The whole muscular wall was destroyed and was replaced by a connective tissue rich in blood vessels. The area proximal to the lumen consisted of connective tissue and of granulation tissue, some

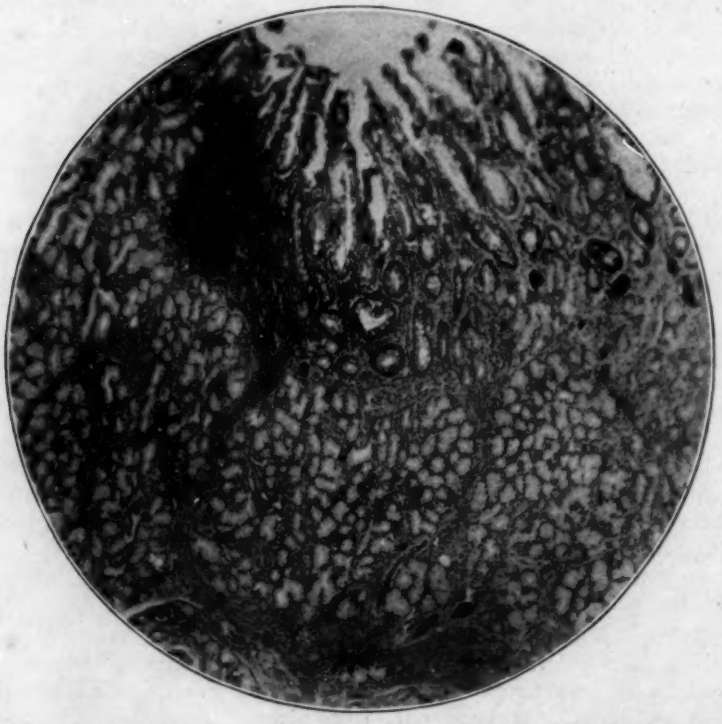

Fig. 1.—Hypertrophic form of gastritis, showing the alveoli with the hypertrophy of the muscularis mucosa growing between the groups of alveoli (pseudopyloric glands of Stoerk); also small islands of intestine-like mucosa with infiltration of round cells.

of which appeared to be new and rich in blood vessels. There were thick-walled arteries and several neuromas in this granulation tissue. There was a necrotic exudate at the base of the ulcer. The adjacent mucosa consisted of branching glands which looked like Brunner's glands, or the pseudopyloric glands described by Stoerk (fig. 1). Parts of the muscle fibers around the ulcer seemed to be destroyed. Two large nerves were found embedded in the muscles. Accumulation of round cells penetrated deep into the muscularis mucosa, and some of the lymph follicles were also found beneath it. In some parts they penetrated through the wall of the glands into the lumen. Small areas of dark-stained epithelium with goblet cells were also found. A conical-shaped defect

separated areas of glands and penetrated into the accumulation of round cells near the muscularis mucosa; on one side remnants of the glands and on the other side dark-stained young epithelium attempted to bridge the defect.

The blood vessels in the scar were thick-walled, some were obliterated, and there were a few islands of round cells and polymorphonuclear leukocytes.

Sections taken through the posterior surface of the antrum, about 5 cm. from the pylorus, demonstrated fan-shaped areas of glands. The foveolae or tubules were straight and long, and the glands were not branching. These looked like normal pyloric glands. Close to the areas just described I found some in which the follicles were increased in number and size; where was also an

Fig. 2.—Islands of dark-stained intestine-like epithelium taken from the same section as figs. 13 and 18, representing a healed erosion.

accumulation and infiltration of round cells between the glands and the muscularis mucosa and between the tubules of the glands. The glands were branching with light-staining epithelium. There were also islands of tubules of dark-staining epithelium, some of which appear high columnar, while others contained goblet cells (fig. 2). In other places, the mucosa sank to the accumulations of round cells and polymorphonuclear leukocytes and appeared to cover the sunken areas by one layer of high columnar epithelium. Where the epithelium reached the accumulation of round cells, it appeared desquamated, and the lymphocytes, leukocytes and plasma cells penetrated through the mucosa into the lumen (fig. 3).

The structure of a section from the anterior wall of the stomach, the same distance from the pylorus, was the same as that of the sections just described,

except that in some parts the tubules or foveolae were not so long, and the glands contained parietal cells. In some areas the accumulations of round cells and lymph follicles occupied a part of the mucous membrane. In certain areas, numerous parietal cells were found in the body and fundus of the glands, in others, only remnants of the foveolae or tubules were left, and the bodies and the fundi of the glands appeared to be replaced by groups of round cells. There were indentations in the mucosa reaching the muscularis. Where the mucosa reached the muscularis mucosae and the groups of round cells, the epithelium was dark-stained and proliferating.

Sections obtained from the proximal end of the posterior wall of the stomach, in the area of the corpus, demonstrated foveolae which were about

Fig. 3.—Small defect in the depth of the glands, healed by single layer of dark-stained epithelium.

one fifth of the length of the glands; these appeared to be typical fundus glands with chief and parietal cells. There were only a few round cells and scanty interstitial tissue between the foveolae and tubules. There was some accumulation of round cells in the places where the mucosa sank down. Here the islands consisted of a few foveolae wide and deep and dark-staining, but glands proper were not seen. In the anterior wall of the stomach, at the area where resection was made, I found the same picture as in the posterior wall, with the exception that there was an area in which the fundus glands appeared to be infiltrated by round cells.

Midway between the pylorus and the resected proximal end, the glands had very deep foveolae admitting many branched alveoli with light-staining epi-

thelial cells. The follicles were greatly increased in size and number and possessed germinal centers; there were many areas of proliferating dark-stained epithelium, also islands of intestine-like muscosa. Some of the foveolae were spread wide apart by an accumulation of lymph follicles, polymorphonuclear leukocytes and plasma cells.

THE NORMAL STOMACH MUCOSA

The mucosa of the stomach is a maze of irregular folds. In the fixed state, there are four longitudinal folds spreading out fan-shaped on the lesser curvature into the pyloric canal. When the stomach is distended, most of the folds disappear. According to Tandler,[8] the part of the stomach containing the Magenblase is free from folds. The color

Fig. 4.—Low power magnification of one mammilla of état mammelonné with axial vessel.

of the normal stomach mucosa is pinkish. Aschoff [9] calls attention to the difference in color between the gastric pathway and the rest of the stomach.

In the condition of distention, when the folds disappear, one can see small areas a few millimeters in width, the areae gastricae, which are indistinctly marked off by irregular sulci. In the large proximal portion of the stomach mucosa of the adult, there are numerous small elevated

8. Tandler, Julius: Lehrbuch der systematischen Anatomie, Leipzig, F. C. W. Vogel, 1923, vol. 11, p. 131.

9. Aschoff, Ludwig: Lectures on Pathology, New York, Paul B. Hoeber, 1924, p. 279.

areas a few millimeters in diameter (fig. 4). This condition has been called, by normal anatomists and pathologists, status mamillaris, état mammelonné or catarrhus verrucosus (see later état mammelonné).

Microscopic examination of the mucous membrane reveals numerous small openings—the foveolae gastricae. These are depressions into which the glands of the stomach open. The foveolae gastricae measure one fourth to one fifth of the depth of the fundus glands. In the region of the pylorus they are much deeper and may even reach the muscularis mucosae. The gastric crypts and folds between them are covered with a long, slender epithelium, the cells of which are somewhat conical. They extend downward in a curve toward the basement membrane, while the broad part of the cell is directed toward the free surface. The latter portion forms a cuplike plug which gives a chemical reaction for mucus, the basal part containing the nucleus being richer in protoplasm.

Oppel,[10] who examined this type of epithelium in vertebrates and mammals, found that, as in man, the cells consist of two differentiated parts—a basal protoplasmic part and a peripheral part belonging to the surface, which he named Oberende. Ebner and Oppel have demonstrated that the open cells found on the surface of the epithelium are artefacts. Cells of the superficial mucosa of the stomach, like other mucous cells, are easily injured. The superficial part of the cell swells and flows in part or altogether from its free end.

The glands of the stomach mucosa are usually divided into the cardiac glands, fundus glands and pyloric glands. Aschoff[9] and Oshikawa[11] have recently called attention to the form of glands in the region of the isthmus, which they named the intermediate glands. There is doubt as to the nature of these glands, and I shall return to them in the presentation of the pathologic picture of the stomach mucosa (see pseudopyloric glands).

The cardiac glands are branched tubular in type and occupy an area varying in width from 0.5 to 4.3 cm. (Bensley[12]). The cells are somewhat similar to those of the pyloric glands and to the mucous chief cells of the fundus glands. Some of the lining cells, however, contain zymogen granules similar to those of the chief cells of the fundus glands. Occasionally a few parietal cells are present.

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10. Oppel, A., quoted by Harri, Paul: Ueber das normale Oberflächen-Epithel des Magens und über Vorkommen von Randsaum-Epithelien und Becherzellen in der menschlichen Magenschleimhaut, Arch. f. Mikrosk. Anat. u. Entwickl. geschichte **58**:684, 1901.

11. Oshikawa: Beiträge zur Histologie des Magens und der Magengeschwüre, Virchows Arch. f. path. Anat. **248**:217, 1924.

12. Bensley, R. R.: Reference Handbook of the Medical Sciences, ed. 3, New York, William Wood & Company, vol. 7, 1917, p. 943.

The fundus glands occupy the larger proximal part of the stomach mucosa. The tubules in these glands are only moderately branched and are vertical. They are close to each other and are separated by a scant amount of connective tissue. Each foveola receives from three to five or more branched tubules. The small part of the tubule opening directly into the faveola is narrow and is known as the neck. The terminal blind end is called the fundus, and the intermediate portion is known as the body of the gland. The glands proper are lined with two kinds of cells, the chief and the parietal cells. The chief cells have been described by Heidenhain [13] and called the Hauptzellen, while Rollett [14] named them Adelemorphezellen. The parietal cells were called by Heidenhain the Belegzellen and Rollett the Delomorphezellen.

The parietal cells are larger than the chief cells; they are more irregular, triangular or oval, and show an affinity for acid stains (fig. 5A). Normally these cells are more numerous in the body and in

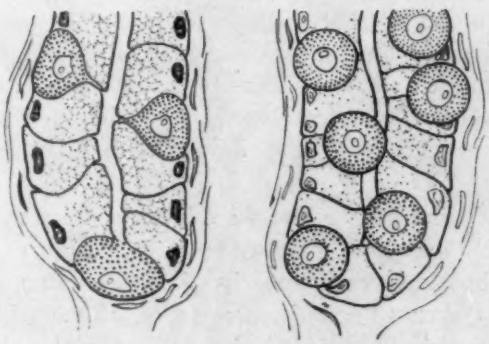

Fig. 5.—*A*, diagram of a normal fundus gland with parietal cells of triangular form; *B*, gland of état mammelonné with round parietal cells.

the neck of the gland. They rest on the basement membrane of the tubule, frequently forming a protuberance beyond the even line of the remaining cells, and occasionally giving the appearance of being extraglandular. They contain one or more nuclei, and with certain stains display intracellular canaliculi connecting the cells with the lumen of the gland by a narrow duct.

The chief cells are wedge-shaped columnar structures which form a lining for the tubule, being displaced in varying degrees by the parietal cells. The chief cells when stained in hematoxylineosin have two well formed zones: a basal deep-staining portion and a superficial light-stain-

13. Heidenhain, R., quoted by Bensley, R. R.: Reference Handbook of Medical Sciences, ed. 3, New York, William Wood & Company, 1917, vol. 7, p. 943.

14. Rollett, A., quoted by Bensley, R. R.: Reference Handbook of the Medical Sciences, ed. 3, New York, William Wood & Company, 1917, vol. 7, p. 943.

ing zone. The nucleus is found at the junction of the two zones and possesses one or two oxyphile nucleoli. The chief cells of the neck are characterized by the absence of chromidial substance and zymogen granules which are in the basal part of the chief cells of the tubule. According to Bensley [12] they are irregular columnar structures and slightly smaller than the chief cells of the body of the gland. The contents of the cell appear clear in hematoxylineosin stain. The nucleus is situated in the proximal attached part of the cell.

These are evidently the cells first described by Zimmermann [15] as possessing a light-staining plasma and a flat nucleus at the bottom of the cell. Zimmermann believed these were mucus-secreting cells similar to those which Kupffer described in the mucus glands of the stomach. According to Bensley,[12] these mucus-secreting cells may be found in a few glands to reach entirely to the bottom and to replace the chief cells throughout the whole gland. Such glands contain a few parietal cells and have a larger lumen.

I am of the opinion that such glands represent the frequent proliferated epithelium of the lacunae which generally displace the specific cells not only in the neck, but also as far as to the base. They represent the glands described by Stoerk [4] (fig. 1), which are characterized by numerous branchings with the lining of cells appearing clear in hematoxylin.

Many histologists believe that there is not a sharp transition between the fundus and the pyloric glands. Radasch,[16] after examining several human stomachs and several stomachs of rabbits, came to the conclusion that there is an abrupt decrease or cessation of the acid cells, and that the branched tubular glands of the pylorus make their appearance suddenly.

In the examination of my own specimens, I have observed that the fundus glands cease abruptly. They are replaced by the branched normal pylorus glands or those demonstrating pathologic changes.

The pyloric glands open into crypts which are much deeper than those of the fundus region. The tubules of the pyloric glands are more branching, less closely set and contain more connective tissue between them. The cells lining the foveolae of the pyloric glands are cylindrical and possess an oval nucleus much like that of the cells found in the bottom of the foveolae of the fundus glands. Those lining the terminal branches of the pyloric glands are more cuboidal, resembling those of the neck of the fundus glands. These are light-staining mucus cells. The parietal cells are either altogether absent or rare.

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15. Zimmermann, quoted by Bensley, R. R.: Reference Handbook of the Medical Sciences, ed. 3, New York, William Wood & Company, 1917, vol. 7, p. 943.

16. Radasch, H. E.: Acid Cells of the Stomach, Internat. J. Gastro-Enterol. **1**:24, 1924.

The foregoing is a description of the normal mucosa. It is important to have a clear idea of what is normal and what belongs to the pathologic. The adult stomach is seldom found to be free from heterotopic or proliferated changes. Orator,[7] who made an extensive study of what he thought were normal and pathologic stomachs, came to the conclusion that the normal mucosa of the fundus region is characterized by foveolae which represent only from 25 to 30 per cent of the whole thickness of the stomach mucosa; that a production of mucus in the fundus glands is slight, and that only in the region of the stomach pits is there a slight cellular infiltration.

Only occasionally is there a lymph follicle with a germinal center in the submucosa. The pyloric glands of the stomach mucosa are characterized by deep foveolae which represent from 60 to 70 per cent of the thickness of the mucosa. These glands secrete mucus in abundance. There is a light cellular infiltration not only in the region of the pit of the stomach, but also in the region of the deeper structure of the glands. In many pathologic stomachs examined, I found areas of mucosa resembling the normal mucosa described in textbooks on histology.

ÉTAT MAMMELONNÉ

I was unable to find any true or comprehensive picture of the condition frequently described as état mammelonné. Aschoff,[17] Borst [18] and other authorities consulted, speak of this condition as catarrhus verrucosus. Aschoff,[17] in his textbook, states that the état mammelonné is found in the region of the antrum pylori. Bassler [19] describes it as representing the rough, mammillated appearance limited to the mucosa of the distal part of the stomach. Tandler,[8] in his recent textbook on normal anatomy, brings out the fact that polygonal areas in the stomach mucosa may be pronounced and elevated, and that the condition is then called status mammillaris.

Stoerk [4] gives a true description of the histologic picture of état mammelonné. He points out that it is not an actual gastritis but a hypertrophy. Many stomachs which I examined present this picture, which is limited to the proximal part. As a rule, the distal part of the stomach shows the microscopic changes of gastritis. The elevations, which cover the mucous membrane of the fundus area of the stomach, are fairly uniform. An inflammatory process is not found microscopically. The

17. Aschoff, Ludwig: Pathologische Anatomie, ed. 6, Jena, Gustav Fischer, 1923, vol. 2.

18. Borst, Max.: Pathologische Histologie, Leipzig, F. C. W. Vogel, 1922, p. 121.

19. Bassler, Anthony: Diseases of the Digestive System, Philadelphia, F. A. Davis Co., 1922, vol. 1, p. 534.

glands are increased in number, and there is an elongation of the gland at its central part. At the base, the gland turns on itself, the elongation of the basal part of the tubule being prevented by the muscularis mucosae. Each mammillated protuberance seems to have an axial vessel (fig. 4). There is an increase in the number of parietal cells, and there are pictures in which the parietal cells overshadow the chief cells (fig. 6). Normally the parietal cells are triangular, oval or wedge-shaped, but in this condition they are round and are found to lie directly

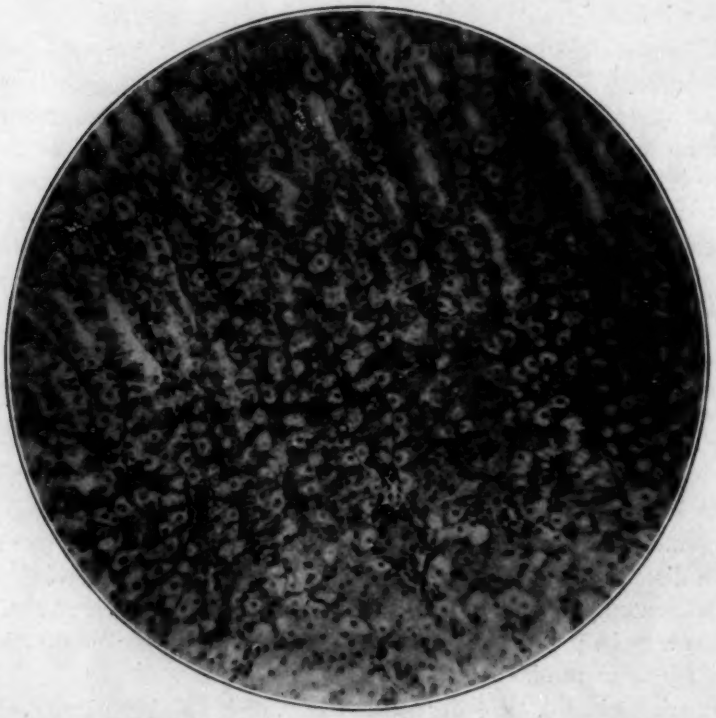

Fig. 6.—Low power magnification of état mammelonné, showing the numerous round parietal cells.

in contact with the lumen (fig. 5B). Droplets surrounding the nucleus can be seen. The parietal cells bulge and may escape, filling the lumen.

In état mammelonné of the stomach mucosa there is little connective tissue between the tubules, and the lymph follicles are few and mostly triangular, a condition which distinguishes état mammelonné typical of the fundus from the condition in the pyloric mucosa, in which numerous round lymph follicles with germinal centers indicate an active inflammatory condition. The areas of gastritis may be interspersed with the areas of état mammelonné in the mucous membrane of the fundus, and occasionally there may be inflammatory changes characteristic of gastritis invading the état mammelonné.

The gastritis which is limited to the distal part of the stomach mucosa is found with about the same frequency as état mammelonné in the proximal part of the stomach mucosa, and both conditions appear to be concomitant in the same stomach. It is difficult to interpret the picture of état mammelonné unless it is considered as a compensatory hypertrophy due to a chronic gastritis in the distal part of the stomach mucosa. Stoerk [4] explains that the chronic gastritis of the antrum may serve as a constant "whip" which stimulates the proximal part of the stomach mucosa (figs. 6, 4, 5 and 7).

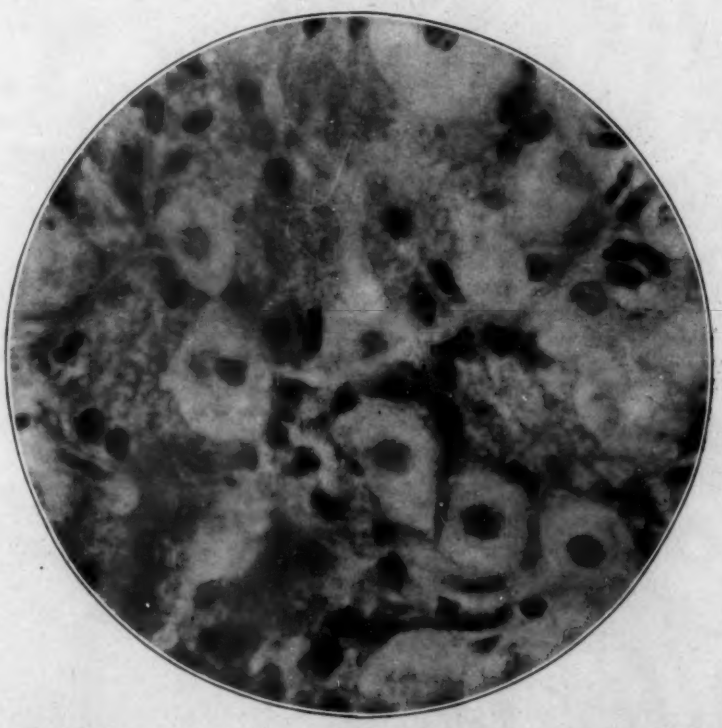

Fig. 7.—High power magnification of état mammelonné showing the irregular parietal cells.

GROSS AND HISTOLOGIC PICTURES OF THE MUCOSA

The gross appearance of the mucosa of the resected stomach varies. The folds may appear less prominent or absent on account of the edematous condition of the mucous membrane (fig. 8). In other specimens the folds of the mucous membrane are pronounced (fig. 9). As a rule, the mucosa of the fundus of the stomach is different in appearance from that of the antrum. The small areas marked off by irregular sulci, which are normally described as the areae gastricae, are much elevated. The elevations found in the fundus as a rule are larger and more nearly equal in size than the elevations which are found in the

Fig. 8.—The distal part of the stomach resected for duodenal ulcer. The mucosa is only partially granular; the granules are not pronounced. Smooth areas of mucosa predominate.

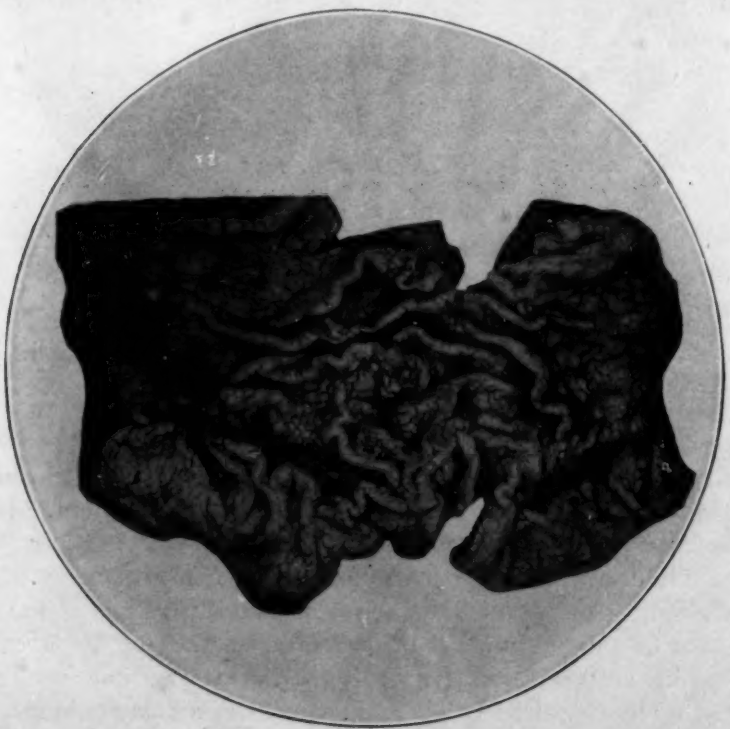

Fig. 9.—Distal end of a stomach resected for duodenal ulcer. Mucosa extremely granular; the granules are irregular in size and shape; smooth areas of mucosa found.

mucous membrane of the antrum; the last are irregular and flat (fig. 9). Such elevations are not always continuous; they may be separated by areas of smooth mucous membrane. Pinkish areas of various sizes, or small pin-point reddish spots may be found in the antrum, while the mucous membrane of the proximal part of the stomach may be pale and retain its system of folds.

Microscopic examination of the antrum mucosa reveals that it is frequently arranged in fan-shaped groups of glands. In some parts the length of the gland is noticeable, and it appears that the mucosa is in a hypertrophic state. In other sections the mucosa appears low, and in still other parts only one layer of epithelium covers the interstitial tissue (figs. 10, 11 and 12). The mucosa may also contain villi-like projections and variously shaped islands of dark-stained epithelium. The lymph follicles appear to be increased in number and size, and there are great accumulations of lymphocytes, leukocytes, eosinophils and plasma cells. In some areas the lymph follicles are either covered by desquamated epithelium or altogether exposed to the lumen of the stomach (figs. 12 and 13).

The surface epithelium may be normal or appear swollen, desquamated with lymphocytes and leukocytes wandering through the epithelial wall. The lacunae are usually deep and wide, giving the appearance of microscopic wells or valleys. These microscopic valleys are more numerous in the region of the antrum and become gradually smaller toward the cardia. Even in the normal mucosa the lacunae are deeper in the region of the antrum than in that of the fundus, but the difference is not so great and the lacunae are not so wide as in specimens examined. According to Moszkowicz,[6] the characteristic depth of the lacunae in the pathologic stomach is not found in the stomachs of children 1 year old. Some of these valleys appear to be dark stained.

On closer examination one sees that the surface epithelium of the dark-stained areas contains numerous mitoses, an indication that the epithelium is in a state of proliferation (fig. 14). Heyrovsky,[3] Hammerschlag[20] and Cohnheim[21] have called attention to this form of epithelium. With the high power lens one finds that the irregular groups or the oval or crescent-shaped islands of the dark-stained mucosa comprise a mixed proliferating type of gland frequently resembling the intestinal epithelium (figs. 1 and 2). The crescent-shaped islands are surrounded by connective tissue, and there are frequently remnants of

20. Hammerschlag: Untersuchungen über das menschliche Magenepithel unter normalen und pathologischen Verhältnissen, Arch. f. Verdauungskr. 2, 1896.

21. Cohnheim, P.: Die Bedeutung kleiner Schleimhautstuckchen für die Diagnostik der Magenkrankheiten, Arch. f. Verdaungskr. **1**:274, 1895.

Fig. 10.—Pronounced gastritis with remnants of glands present and groups of round cells. Low power magnification.

Fig. 11.—An extreme degree of gastritis, with only one layer of desquamated epithelium covering the interstitial tissue. Low power magnification.

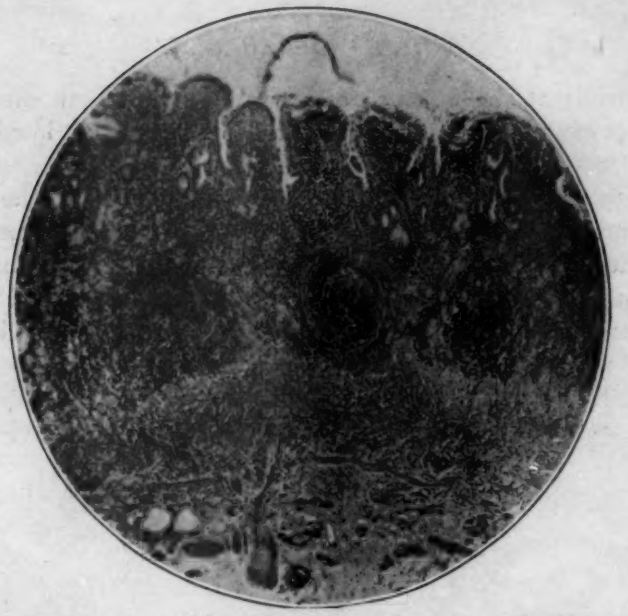

Fig. 12.—A form of gastritis with accumulation of round cells and increase in number and size of lymph follicles (follicular gastritis). Low power magnification.

Fig. 13.—From the same section as figure 18. The progressive destruction of the glands and the adenoid tisues. Plug of mucus covering the defect (follicular ulcer).

normal glands at the bottom of these islands. I shall discuss the significance of the islands of heterotopic or atypical glands in connection with the erosions.

There is a prevailing form of gland which Stoerk called the pseudo-pyloric type. The tubules are pushed apart by interstitial elements with lymphocytes and leukocytes. The fundi of the glands are branching and irregular. Muscle fibers from the muscularis mucosa and the connective tissue penetrate deeply between the glands. The cells lining the

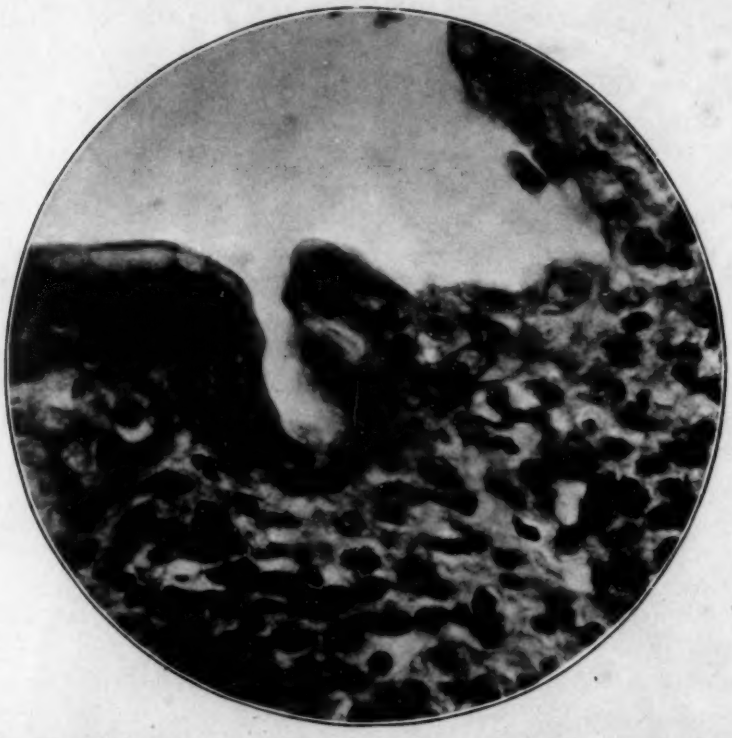

Fig. 14.—High power magnification of figure 17 to demonstrate the dark-stained syncytium-like area of epithelium attempting to heal the defect.

branching alveoli are transparent and cuboidal with the nucleus in a marked basal position. One frequently finds that the lymph follicles are numerous and large and possess a germinal center in the region of these glands. This form of gland is not only found in the antrum mucosa, but may also be found remote from the pylorus in the fundus of the stomach displacing the typical glands. The lumina of such glands are occasionally filled with lymphocytes, leukocytes and plasma cells (fig. 1).

Oshikawa [11] and Aschoff [9] have described a new form of gland found in the isthmus of normal stomachs, which they named the intermediary

gland. Such glands, according to these authors, are almost entirely lacking in chief cells, but contain the parietal cells in varying numbers. The epithelial cells lining these glands, the intermediary or adjacent cells, resemble the epithelial cells of the pyloric glands. Aschoff is of the opinion that the isthmus or intermediary glands described by himself and Oshikawa are the same glands which Stoerk [4] designated as the pathologic or pseudopyloric glands. One frequently finds the type of gland described by Stoerk. The presence of increased intracellular elements, the increase of lymph follicles in size and number and the presence of undifferentiated islands of regenerated epithelium gives the impression that such glands are atypical. These glands are also found in the fundus mucosa replacing the areas of état mammelonné when there is an extensive gastritis involving the greater part of the stomach. This supports Stoerk's opinion that they are a type of pathologic glands.

Microcysts surrounded by a single layer of epithelium and atrophic areas of mucosa are also to be observed in the sections.

INTESTINAL TYPE OF EPITHELIUM

Various authors have called attention to the finding of an intestinal type of the epithelium in the normal and pathologic stomach. According to Müller, Weber was the first one to call attention to "some other glands which often belong to the same categories of the usual mucous glands same as Lieberkühn's glands. Härri,[22] A. Schmidt,[23] Hammerschlag,[20] Boekelman,[24] Heyrovsky,[3] Moszkowicz,[6] Stoerk [4] and others have called our attention to the same observations. One does not experience difficulty in finding intestine-like mucosa in the specimens examined. It is a common observation. Intestine-like mucosa is easily recognized at the first glance, since the goblet cells and the Paneth cells containing acidophilic granulations break the monotony in the midst of other components of the stomach mucosa. The intestinal glands may be circumscribed, diffuse or in the shape of a crescent, giving the impression of islands among mucous membrane otherwise uniform in appearance (figs. 1, 15 and 2). Occasionally the areas of the intestinal type are much larger and may be isolated or in groups and overshadow the normal structures.

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22. Härri, Paul: Ueber das normale Oberflächen-Epithel des Magens und über Vorkommen von Randsaum-Epithelien und Becherzellen in der menschlichen Magenschleimhaut, Arch. f. Mikr. Anat. u. Entwick, **58**:684, 1901.

23. Schmidt, A.: Untersuchungen über das menschliche Magenepithel unter normalen und pathologischen Verhältnissen, Arch. für path. Anat. u. Phys. **143**:477 (March 9) 1986.

24. Boekelman, W. A.: Untersuchungen zur pathologischen Anatomie des menschlichen Magens in Fällen von Ulcus und Carcinom bei bekannter chemischer und motorischer Funktion, Ztschr. f. klin. Med. **44**:128, 1902.

On closer examination one finds that the cells possess a cuticular border. The cells with acidophilic granules are situated at the bottom of the tubules. The mucosa of the stomach epithelium stains red with the acid components of the Jenner stain, while the goblet cells stain blue with the basic elements of the stain.

There seems to be a dispute as to the true nature of these observations. The majority of investigators agree that such heterotopic intestine-like pictures are a result of the various destructive and pathologic processes of the stomach mucosa. Such islands are frequently sur-

Fig. 15.—Two islands of intestine-like mucosa found in otherwise normal pyloric mucosa. Low power magnification.

rounded by atypical glands. Hammerschlag,[20] Heyrovsky,[3] Stoerk [4] and Moszkowicz [6] believe that the aberrant islands in the mucous membrane are produced by pathologic processes. The adjacent epithelium covers the defect with an indefinite kind of epithelium which later gives rise to the stomach or intestinal type of epithelium.

Müller,[25] however, maintains that intestinal islands are due to disturbed embryologic development. He could not demonstrate any

25. Müller, P. F.: Beitrag zur Histologie und Parhogenese des Ulcus peptic um ausserhalb und innerhalb des Magens, Beitr. z. klin. Chir. **123**:1, 1921.

transition from one kind of epithelium to another. Stoerk [4] demonstrated intestinal islands of epithelium in a scant and solitary fashion in the pyloric region of the new-born; he believed that the observations there were congenital and represented a real type of heteroplasia.

The intestine-like mucosa in adult stomachs cannot, however, be explained on the same basis. The cells of the stomach epithelium are constantly being replaced by new epithelium. There is reason to believe that the embryonic cells are also shed by the time the individual has reached the adult age. Stoerk [4] is of the opinion that the intestinal islands in the adult are a result of gastritis, and that they remain as a mark of former destruction of mucous membrane. This can perhaps be explained by the fact that in the early fetal state the gastro-intestinal tract is lined by a uniform type of epithelium which forms both intestinal and stomach epithelium. Such possibilities lie dormant in the stomach epithelium and are awakened only under the influence of irritation or vehement inflammation. These powers are awakened in the undifferentiated type of epithelium which attempts to close a defect.

According to Fischel,[26] the reversion to type is accepted not only as possible but as a condition on which the possibilities of regeneration are dependent in the lower animals. The process of reversion is necessary in order to rejuvenate the cells and enhance their power of differentiation. Moszkowicz [6] thinks that the process of differentiation of the epithelium depends on reaction to changed conditions of environment. Normal development and differentiation are also produced under the influence of certain stimuli which the tissues exert on each other. When these stimuli disappear a reversion process is produced, the end-results of which resemble the developing stage of the organ or tissue. The original embryonic potential powers to produce earlier cell forms may therefore be lost.

Moszkowicz,[6] quoting a personal communication from Fischel, states that the development of the human ovum takes place in such a manner that the various parts are not only able to give rise to normal developing tissues, but are also able to produce other tissues when the normal process of development is destroyed. Concomitant with the progress of differentiation, there is a diminution in the potential powers of differentiation. The power of the cells to produce atavistic tissue is reduced in proportion to the state of its highly developed function. The original germ layers possess many potential powers and can form many kinds of tissue. Experimentally, Fischel [26] found that these potential powers stop at a certain stage when the ectoderm can produce only ectodermal tissue and the entoderm only the entodermal tissues, etc. The potential

26. Fichel, Alfred, quoted by Moszkowicz, Ludwig: Zur Histologie des ulcusbereiten Magens, Arch. f. klin. Chir. **122**:444, 1922.

powers of the germ layer are more limited than those of the original ovum, and they become diminished with the gradual differentiation of the organ. Under normal conditions, the adult grown cell produces its own kind, while under experimental conditions, the dormant powers of the cell will have a wider scope.

The cells in the glands of the stomach mucosa are destroyed by various agents. The defects are healed by a rapid cell proliferation. Through the loss of connection with the surrounding old cells, new mechanical or chemical conditions are produced which have a tendency toward nondifferentiation. Under new conditions and new environment, the cells may demonstrate new possibilities through the awakening of dormant powers. In the crypts and tubules which have dark-stained epithelium the protoplasm and nuclei frequently have the appearance of a mass where the cell border may be absent and only nuclei are seen. In such groups of cells and nuclei there are groups of regenerated epithelium, goblet cells, Paneth cells and cells with cuticular borders.

EROSIONS

Microscopic ulcerations and defects in the mucosa were encountered in all the stomachs I examined. Some of these defects were funnel shaped (fig. 13) or resembled the structures of a chronic ulcer (fig. 16). Others had bizarre forms (fig. 17) and displaced only one or two tubules or only one foveola. Some extended to the muscularis mucosa, while others involved only the superficial part of the mucosa. Not a few of these ulcerations were in close relation to the many lymph follicles and the groups of round cells (figs. 18 and 3).

The adjacent epithelium at the edge of the defect either looks normal or is swollen, desquamated and mixed with leukocytes and lymphocytes. Even with low power magnification one frequently observes that the epithelium at the edge of the defect is stained darker than the rest. With high power magnification one sees that this epithelium shows definite evidence of regeneration and proliferation, not unlike the process seen in the healing of a chronic ulcer (figs. 17 and 16). The epithelium is seen to be fused in buds with numerous nuclei and a few polymorphonuclear leukocytes (fig. 14).

The funnel-shaped ulcerations are filled with mucous plugs or exudates containing leukocytes, lymphocytes and desquamated epithelium (figs. 18 and 13). At the base of an ulcer occasionally there is necrosis of tissue, and there is evidence of granulation tissue below. The defects are surrounded by glands, some of which appear normal, while others are branching, lined with an epithelium resembling that of the pyloric glands. The glands are directed either obliquely or perpendicularly toward the lumen of the defect. A rolling-in of the fan-shaped groups of glands toward the defect is also observed.

Fig. 16.—Microscopic ulcer from the antrum showing all the histologic characteristics of an old chronic ulcer.

Fig. 17.—Conical erosion demonstrating attempts at healing by dark-stained epithelium.

There are many depressions lined by a single layer of epithelium and islands of dark-stained glands which are of the same shape as some of the erosions described and which give evidence of healed defects (fig. 2). In certain sections of the stomach mucosa one finds funnel-shaped and crescent-shaped islands which contain atypical elements and which are separated by connective tissue from the glands around them. Such islands show evidences of proliferation and frequently contain goblet cells and Paneth cells. Stoerk [4] and Moszkowicz [6] have pointed out the relationship of such islands of intestine-like mucosa and other atypical,

Fig. 18.—Beginning of breaking down of the glands and adenoid tissue.

dark-staining, proliferating glands to the erosions. One frequently sees such an island with glands branching toward the muscularis mucosa from one point at the surface. The epithelium of such an island may be uniform, dark-stained, cuboidal or cylindrical, and may also resemble the epithelium of the intestine.

I have noticed such pictures in the same section with erosions in process of healing, with regeneration of the epithelium in various stages. I believe that the islands of atypical mucosa indicate a healed process of a previous destruction of the mucosa (figs. 2, 13 and 18 are taken from the same section and represent erosions in process of formation and healing).

I have already mentioned that accumulations of round cells and follicles are exposed to the lumen of the stomach where the covering is either in a single desquamated layer or absent. It appears that there is some relationship between the formation of some of the erosions and the presence of lymph follicles and the accumulation of round cells. The wandering of lymphocytes through the covering of the epithelium of such lymph follicles is occasionally observed. Heyrovsky [3] is of the opinion that there is a possibility that the lymph follicles give rise to ulcerations. Moullin [27] believes that the etiologic factors of the erosions and duodenal and gastric ulcers are to be found in the lymph follicles. Konjetzny [5] thinks that the broken down lymphocytes and leukocytes set free tryptic enzyme, which, together with the gastric juice, may penetrate the epithelium which has previously been weakened by an inflamed process.

One frequently observes lymph follicles and groups of round cells lined by a single layer of epithelium. It is difficult to state whether such a single layer of epithelium is a result of a healed process. If a single layer of epithelium normally lines a lymph follicle, it could also easily be injured by the gastric juice allowing the cells of the lymph follicle to escape.

It is questionable whether this group of defects in stomachs resected for various pathologic conditions should be classed with the erosions found in postmortem examination and described in the various textbooks of pathology. Aschoff [17] and Hayashi [28] have pointed out that the erosions found post mortem or in experimental animals are of two kinds: They are the stigmas of Beneke or the small hemorrhagic erosions which are found all over the fundus mucosa, especially on the greater curvature, and the larger, nonhemorrhagic, fresh erosions found in the region of the gastric pathway.

The small hemorrhagic erosions have been called the fundus erosions by Aschoff.[17] They are genetically related to the small wedge-shaped hemorrhages of the stomach mucosa. The base of these wedge-shaped hemorrhages corresponds to the surface of the mucous membrane, and the apex is directed toward the muscularis mucosa. The defects found in the stomach resected for various surgical conditions are not uniform in size or shape; they may be funnel-shaped, crescent-shaped or irregular.

There has been much discussion concerning the etiology of the hemorrhagic erosions. Aschoff [17] has observed that such erosions arise after repeated vomiting. He thinks that the blood from the portal system cannot pass the liver fast enough during the act of vomiting,

27. Moullin, C. M.: The Essential Cause of Gastric and Duodenal Ulcer, Lancet, p. 993, Oct. 1, 1910.

28. Hayashi, T., quoted by Oshikawa: Beiträge zur Histologie des Magens und der Magengeschwüre, Virchow's Arch. f. path. Anat. **248**:217, 1924.

when the abdominal muscles and the diaphragm are responsible for the compression of the viscera. The recoil waves of the blood in the portal circulation are stopped when the blood in the capillaries of the portal system meet the arterial capillaries close under the mucous membrane. The capillaries burst, and a hemorrhage is produced. Beneke,[29] however, is of the opinion that such primary hemorrhages are not of importance in the formation of hemorrhagic erosions as generally understood.

The embolic theory has been emphasized by many European and American authors from the pathologic observations and experimental work. Vasomotor changes of the arterioles have been considered responsible for such hemorrhages. Hemorrhages and erosions have also been produced experimentally through the spasm of the muscularis mucosa by the injection of pilocarpine. There are many other experimental methods by which hemorrhagic erosions can be produced.

The etiologic factors of the erosions found post mortem, or produced experimentally, are closely related to the agonal phenomenon of death. Just before death, when the muscle of the heart becomes weak, and its action such that the circulation in the stomach mucosa is slower and weaker, a rapid focal digestion may take place, causing erosions. The various toxins of infectious diseases causing parenchymatous changes in the other organs of the body may, in the same manner, easily disturb the protective agency of the stomach mucosa against self-digestion, and also produce various hemorrhagic erosions.

The erosions and defects described are found in the mucosa, which is subject to gastritis in its various manifestations. I could not find any changes in the blood vessels or hemorrhages in the mucosa which would explain the etiology of these defects. Nauwerck[30] and Konjetzny[5] have also failed to find any changes in the blood vessels, such as endarteritis, sclerosis, emboli or thrombosis, which would explain the acute formation of these erosions. The histologic picture of the defects found in the surgical material concomitant with the pathologic condition of the rest of the stomach mucosa makes these defects and erosions different from the hemorrhagic erosions found post mortem.

THE NATURE OF INFLAMMATION

The nature and significance of the histologic picture and the factors concerned in the production of such frequent changes in the mucous membrane of the stomach are still problematic. The gastritis is evidently

29. Beneke, R., quoted by Aschoff, L., Pathologische Anatomie, ed. 6, Jena, Gustav Fischer, 1923, vol. 2, p. 715.

30. Von Nauwerck, C., quoted by Konjètzny, George Ernest: Chronische Gastritis und Duodenitis als Ursache des Magenduodenalgeschwürs, Beitr. z. path. Anat. **71**:595, 1923.

not a result of actual invasion of bacteria, since there is seldom a typical picture of bacterial inflammation or the type of inflammation that is usually seen in the mucous membrane of other organs. This form of gastritis is characterized by a proliferative and regenerative phenomenon resulting in the pseudopyloric glands of Stoerk, the intestinal islands and the dark-stained groups of epithelium, formation of microscopic ulcerations and increase in the lymphoid elements.

Usually the repair of injuries is characterized by the formation of intensive blood supply, granulation tissue and the elimination or absorption of the injurious substances, and later by an attempt at restoration to normal. Marchand [31] points out that an attempt of the organ to repair loss of tissue and to regenerate is not always accompanied by inflammation. The process of inflammation is developed only in the higher vertebrates, and even among these there are definite differences. In the lower animals the defense against injury is accomplished only through the process of regeneration. The same process takes place in the early stages of the human embryo, and only in its later stages does inflammation occur.

In the stomach mucosa an infiltration of leukocytes and an increase in the number and size of the lymph follicles are observed; however, as Moszkowicz has pointed out, these changes are not always related to the erosions and ulcerating areas or the islands of heterotopic epithelium, and even in chronic callous ulcers there are only a small wall of leukocytes and edema. The reaction of defense does not appear to be commensurate or significant in comparison with the reaction of the same insults to the mucous membrane of other organs. Moszkowicz believes that gastric juice is directly responsible for the inhibitory influence on the inflammatory process. The formation of granulation and scar tissue takes place when the muscularis mucosa is broken through, but even here the gastric juice prevents any extensive formation of the components of inflammation. Only when the liver, pancreas or other organs in the abdomen are near an ulcer does an inflammatory peritonitis prevent a sudden perforation of the ulcer. When the ulcer faces the free abdominal cavity, there is often a perforation without previous symptoms because there has not been sufficient inflammatory reaction.

It is possible that the factors which cause the changes in the stomach mucosa are of a protean nature. Acute gastritis produced in the course of an infectious disease through the action of bacterial toxins, or through a direct bacterial invasion of the wall of the stomach, may be the beginning of chronic gastritis. In the discussion on the erosions I have men-

31. Marchand, F.: Der Process der Wundheilung mit Einschluss der Transplantation, Deutsche Chirrugie, Stuttgart, Ferdinand Enke, 1901.

tioned the pepsin-antipepsin balance, which is easily disturbed and which contributes to the various acute lesions of the stomach mucosa. Repeated chemical, thermal and mechanical injuries to the mucous membrane through indiscretions of diet, causing the various disorders of the stomach in infants and transitory dyspepsias in adults, are considered to be a starting point in the production of the histologic chronic gastritis. The mechanical, thermal and chemical insults may not be extensive, but if repeated will eventually terminate in a chronic gastritis. According to Stoerk,[4] chronic gastritis can be produced only by various insults of the stomach mucosa through the innervation peculiar to the stomach, since the repeated injuries are not extensive.

When the wall of the stomach of an experimental animal is stimulated in one part, even if the omentum is pinched by an artery forceps, the whole wall becomes pale. Occasionally a depression can be seen in the wall of the stomach after the omentum is pinched. Stoerk believes that it is this peculiar innervation through which an injury in one part of the stomach, bringing other parts of the stomach into reflex and sympathetic stimulation, may disturb the secretory innervation and injure the pepsin-antipepsin balance. Limited and repeated injuries to the stomach mucosa can produce an extensive gastritis through a process of multiplication of the injury connected with its peculiar innervation.

COMMENT

The pathologic changes observed in the mucosa of the antrum reach higher along the lesser curvatures of the stomach. Some of the valleys are covered by only one layer of epithelium, which is frequently desquamated or destroyed. The fan-shaped groups of glands bend over so as to cover some of the walls almost completely. It is possible for the gastric juice to be held in these depressions for a longer period than usual, which leads to desquamation or ulceration of the mucosa.

The various microscopic erosions and defects found in all stages of destruction and healing do not resemble the typical fresh hemorrhagic erosions, the so-called stigmas of Beneke and the experimentally produced erosions. In this connection a more critical interpretation must be made of the experimental work as related to focal infection as the etiologic factor of ulcer of the stomach or duodenum. I have pointed out the vulnerability of the stomach mucosa, the agonal factors of death contributing to the formation of hemorrhagic erosions and the numerous agents which have been employed in experimental work and which have been successful in producing stomach erosions.

I was impressed with the ulcerations and defects found in the stomach mucosa of the surgical material examined as closely related to the concomitant gastritis found in these stomachs. I was not able to

find any emboli or thrombi or any further evidence of disease in the smaller blood vessels near the ulcerations. It is still a debatable point which is primary, gastritis or the erosions and ulcer. Nauwerck [30] and Konjetzny [5] are of the opinion that ulcer is secondary to gastritis. Hauser,[32] Hemeter [33] and Heyrovsky [3] have indicated that gastritis is secondary to ulcer. Konjetzny, on examination of several hundred specimens, has repeatedly called attention to the small erosions of the stomach which are found concomitant with ulcer of the stomach and duodenum and which are in the various stages of healing. He believes that the erosions change into chronic ulcers under the influence of functional and chemical factors.

Many investigators now agree that a chronic ulcer originates in a small erosion. Most of the erosions heal, with the exception of those found on the lesser curvature at the pylorus or at the isthmus of the stomach, which become chronic ulcers. Aschoff [9] has called attention to the anatomic and physiologic factors as related to the production and chronicity of the ulcers along the lesser curvature. He believes that the spasm of the narrow pass and the highway of the stomach are the anatomic and physiologic contributing factors. He also points out the deficient blood supply of the lesser curvature.

I have frequently observed in the specimens examined that when cancer does not originate in a chronic ulcer, it is closely associated with a peculiar heterotopic and hypertrophic condition of the glands of the stomach. The glands have lost their resemblance to the original fundus or pyloric glands. The tubules and alveoli are increased in size and number. The glands are heterotopic and irregular, with a few microcysts present. The cells lining the tubules are large, definitely high columnar, light staining with basal nuclei (fig. 19). It is difficult to state whether such disease pictures represent a hypertrophic form of gastritis or definite cancer. It is my opinion that such a hypertrophic mucosa represents a precancerous condition, since I find concomitant with such pictures definite cancer, tubular or of a simple infiltrating type (fig. 20). I hope to consider this phase of pathologic conditions of the stomach in another article.

I wish to emphasize the division of the stomach into two parts, a fact which is of etiologic interest and worthy of consideration from a therapeutic point of view. The pars digestoria, which is the largest proximal part of the stomach, makes only tonic contractions, and is lined by

32. Hauser, Gustav: Des chronische Magengeschwür, Leipzig, F. C. W. Vogel, 1883.

33. Hemeter, John C.: Experimental Basis of the Dietetic and Medicinal Treatment of Hyperacidity and Gastritis, J. A. M. A. **29**:196 (Oct. 9) 1897.

Fig. 19.—From the same specimen as figure 20, showing the proliferating mucosa at the ulcerated edge of the cancer. (It is not possible to state definitely whether this is a proliferating type of gastritis or actual cancer.)

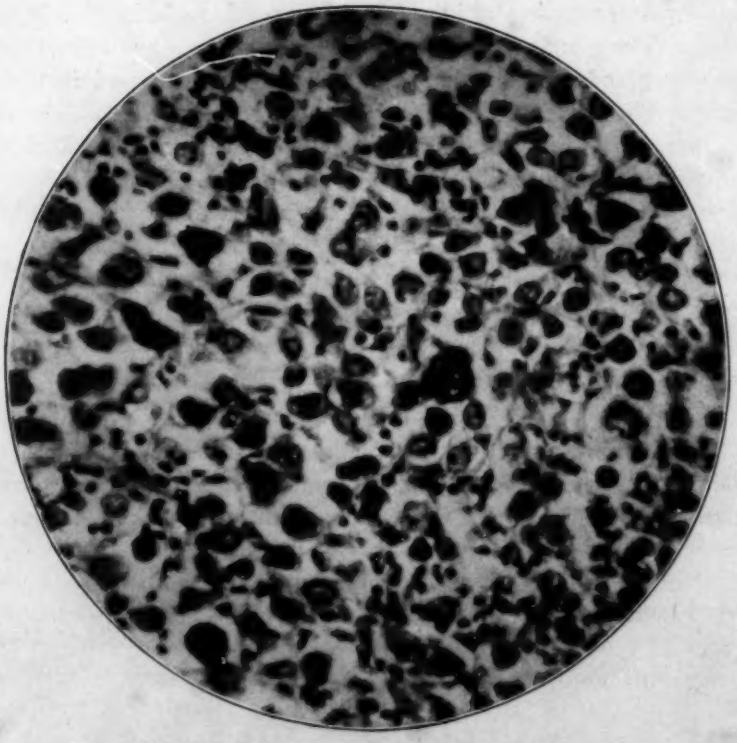

Fig. 20.—Infiltrating simple type of cancer.

fundus glands which are frequently hypertrophic (état mammelonné). The fundus glands, however, are resistant to disease. Through the pars egestoria, or distal part, pass the active peristaltic waves which propel the food into the duodenum. It is lined by less differentiated mucosa which is not as resistant as the mucosa of the fundus glands and which is most frequently subject to the various forms of gastritis. It is also in this part of the stomach that ulcers and cancer are found.

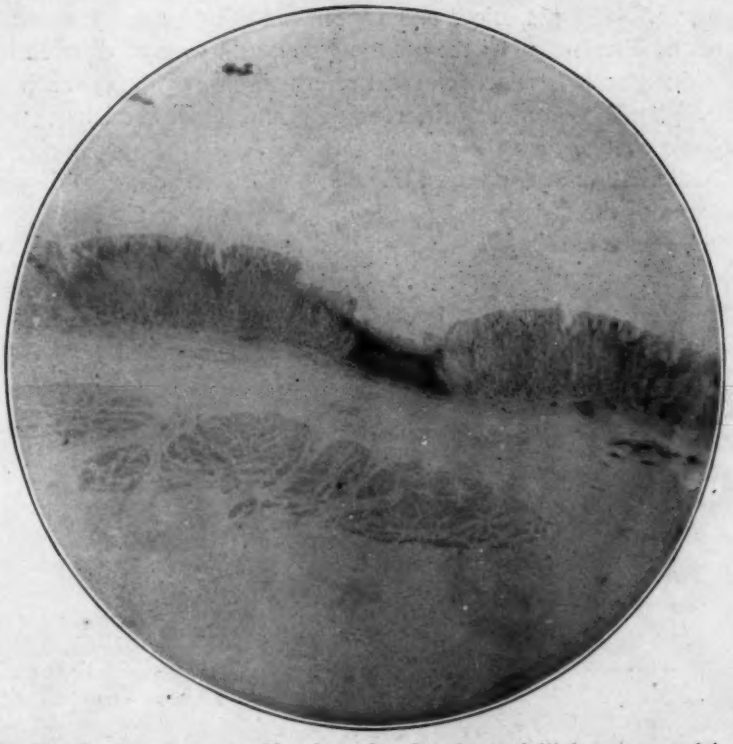

Fig. 21.—Low power magnification of a flat, large follicle uncovered by epithelium (follicular erosion).

SUMMARY

The histologic examination of twelve stomachs resected for ulcer of the duodenum or ulcer of the stomach and eight stomachs resected for cancer reveals that there are evidences of gastritis in the mucosa of all stomachs examined. This form of gastritis is characterized by islands of intestine-like mucosa, areas of dark-stained epithelium with evidences of regeneration and glands with numerous branching alveoli lined with a light-staining cuboidal epithelium (pseudopyloric glands of Stoerk), increase in size and number of lymph follicles and other lymphoid elements. Defects in the mucosa, erosions and small microscopic ulcers are frequently encountered.

The changes are mostly limited to the distal part of the stomach in the area of the distribution of the pyloric glands. The mucosa of the fundus displays a form of hyperplasia and hypertrophy with increase in the size and number of parietal cells described as état mammelonné. Contrary to the opinions and descriptions found in textbooks on pathology, état mammelonné is limited to the fundus mucosa and is not an inflammatory process.

The evidences of gastritis are more pronounced in specimens resected for cancer. Gastritis, ulcer and cancer are found most frequently in the area of distribution of the pyloric glands; the area of distribution of the fundus glands is seldom the seat of any disease of the stomach. The fundus glands are more differentiated and more resistant to any pathologic change than the pyloric glands.

The limitation of the varied diseases of the stomach to its distal part in the area of the distribution of the pyloric glands should be of interest and importance in relation to the surgical prophylaxis and surgical therapeutics of cancer and ulcer.

FRACTURES OF THE SKULL

COMPLICATIONS AND CAUSES OF DEATH: A REVIEW OF 512 NECROPSIES AND OF 61 CASES STUDIED CLINICALLY

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Part I. Complications Causing Death.
- Description of the author's material.
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Recovery under expectant treatment.
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Summary.

The complications which result from fractures of the skull can be placed in three different categories, namely: (1) those that are inevitably fatal, (2) those for which the patient can be treated successfully by craniotomy and (3) those that heal spontaneously if treatment is not given.

In order to study the fatal cases, 512 cases which came to necropsy are here reviewed. Sixty-one clinical records are also presented so that the effects of craniotomy and the final results of expectant treatment may be considered.

PART I. COMPLICATIONS CAUSING DEATH

DESCRIPTION OF THE AUTHOR'S MATERIAL

The 512 necropsies referred to in this article were performed by me or were seen in my work as Assistant Medical Examiner of New York

TABLE 1.—*Age Incidence and Casualties in 507 Necropsies*

Age	No. of Cases	Age	No. of Cases
1–10	46	50–60	87
10–20	20	60–70	58
20–30	63	70–80	25
30–40	90	80–90	2
40–50	115	90–100	1
	Casualty Incidence		
Automobile accidents	180	Knocked down in assault	11
Street car accidents	19	Horse kick	1
Bicycle accident	1	Struck by blunt instrument	21
High falls	41	Hit by falling object	5
Low falls	61	Hit by flying object	1
Fall down stairs	51	Hit by moving object	3
Thrown down in assault	3	Nature of casualty unknown	100

City between Jan. 1, 1920, and March 15, 1926. Five cases of the entire number were those of infants under 5 years of age, and these are considered separately. The remaining 507 cases included persons of all ages and conditions. Of this number, 445 were men and sixty-two women, which is the proportion to be expected in cases of injury. Taken by decades, the age incidence showed that the middle period of life is the one in which fracture is most likely to be included. The figures for the different ages are given in table 1. Highway accidents, which included automobile, street car and bicycle accidents, formed over 40 per cent of all the cases. The fracture was generally produced by the vehicle striking the person's head directly, by knocking his skull against the street or by crushing his head in some way.

The other types of injury were caused by the head striking against a solid surface, or were produced by some object coming into violent contact with the head. The first group included falls or assaults in which

the deceased was either knocked or thrown down. The second group comprised assaults by blunt instruments, kicks by horses, blows and falling objects of any kind striking the head.

In 100 cases the cause of the fracture was unknown, as the deceased was found either dead or in a coma. Most of these injuries were probably caused by falls.

CLASSIFICATION OF THE FRACTURES OF THE SKULL

It is necessary to classify fractures of the skull according to their location, form and extent, because these features generally suggest the intracranial complications that are likely to be present. Any attempt of this kind, however, is difficult, as the fractures cannot be separated easily into definite, well marked categories. Many transitional forms occur which are hard to fit into any scheme of classification.

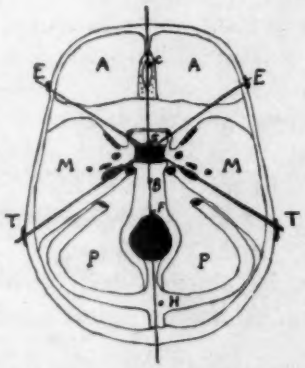

Fig. 1.—Diagram of the base of the skull showing its separation into fossae and how the boundaries of the fossae converge toward the body of the sphenoid bone. *A* represents the anterior fossa; *M*, middle fossa; *P*, posterior fossa; *E*, location of the external angular process of the frontal bone; *T*, location of the mastoid process of the temporal bone; *C*, crista galli of the ethmoid bone; *S*, body of the sphenoid bone; *B*, basilar process of the occipital bone; *F*, foramen magnum; *H*, torcular Herophili.

A preliminary consideration of the anatomic characteristics of the skull and the way in which different fractures are produced, however, is helpful in attacking the problem.

First, the skull must be regarded as an oblong box composed of a vault and base. The vault is a strong, curved dome of bone, of even thickness, with the exception of two fragile plates in each temporal region. The base, however, is an irregularly flattened structure, composed of heavy masses of bone, weakened by thin plates, numerous foramina and bony cavities. The strong and weak points in the base, furthermore, have a systematic arrangement which is best illustrated by a diagram (fig. 1).

The base shows a division into six fossae, two anterior, two middle and two posterior. A strong mass of bone, the external angular process of the frontal, separates the outer extremity of each middle fossa from the anterior fossa in front. In the same way, the mastoid and petrous processes of the temporal bone separate the middle and posterior fossae. Strong structures of bone also come between the two anterior and the two posterior fossae. As figure 1 indicates, the body of the sphenoid is the common center or hub which connects these masses by long ridges, forming a radiating figure with six spokes.

The bone between the spokes forms the floor of the different fossae, and is generally thin, especially over the orbital roof and middle ear cavity. The spokes are, however, rather strong and tough, except at the different points of weakness in the anterior and posterior boundaries of the middle fossae, of which the sphenoidal fissures and the ear cavities are the most important. The central point of the skull is also rendered less resistant by numerous foramina and cavities. The alternating areas of strength and fragility have an important influence on the course of the fracture lines in the skull.

The fractures are almost always produced by the application of violence to the vault, which causes the skull to change its shape, so that the bone becomes especially flattened at the site of impact. The fracture lines generally start at this point, as it is here that the greatest strain is thrown on the continuity of the cranial bones.

The fractures follow certain well defined rules. In most instances they run from the vault down into the base, though sometimes they travel along the vault instead. In the base, the fracture lines generally select the bed of the fossae between the strong masses of bone that form its boundaries and, for the most part, are directed toward the central point of the skull. The fractures also tend to run in the direction of the line of violence. When the point of impact is in the back of the head, the direction is from the back to the front. When the force strikes on the side of the head, the fracture runs toward the opposite side, and if the contact is in the frontal region, the fracture is directed posteriorly.

In general, the crack in the bone has a tendency to seek the lines of least resistance. An expanse of skull with gradual curvature is less resistant to violence than an area of bone with sharp curvature, and the fracture most often seeks out the former unless other factors are present which modify its course. The normal suture lines of the skull, especially on the vault, are also involved in many fractures because they are naturally weaker than the rest of the bone.

The most convenient classification of fractures is into the two main categories of: (1) those involving both vault and base, among which are included a few confined to the base alone, (2) those involving only the vault.

The first category was further subdivided according to the distribution of the fractures into: (*a*) posterior fractures; (*b*) lateral fractures, and (*c*) anterior fractures.

A further subdivision was made, according to the form and extent of the fracture, into: (*a*) linear or fissure fractures, single linear cracks in the bone, showing at the most only a few minor side branches; (*b*) composite fractures or fractures composed of several fissures. The milder forms showed only a few fracture lines running in the same direction as the line of violence. Others showed extensive comminution of the bone at the site of injury.

The vault fractures were subdivided into: (*a*) linear fractures; (*b*) composite fractures, and (*c*) depressed fractures. The last named category was identified whenever the bone was broken into several plates and depressed inward in a limited, sharply defined area of the vault. The arrangement of the material in this classification is shown in table 2.

TABLE 2.—*Kinds of Fractures Studied*

Fractures involving vault and base			471
(1) Posterior location		178	
(a) Linear fractures	96		
(b) Composite fractures	82		
(2) Lateral location		232	
(a) Linear fractures	99		
(b) Composite fractures	133		
(3) Anterior location		61	
(a) Linear factures	28		
(b) Composite fractures	33		
Vault fractures			36
(a) Linear fractures		12	
(b) Composite fractures		16	
(c) Depressed fractures		8	

Fractures Involving Vault and Base.—(1) Posterior Location: The posterior fractures were relatively numerous; they occurred in about 35 per cent of the entire number of cases. They all followed an impact in the posterior region of the vault which caused shortening of the sagittal diameter of the skull. The lines of fracture traveled down into the posterior fossae in the majority of instances, although some also extended into other regions of the skull (figs. 2 and 3).

(2) Lateral Location: As the sides of the vault are the most expansive areas in the cranium, and as the bone is the flattest and most exposed to the trauma, it is not surprising that lateral fractures reached the proportion of 45.9 per cent. They were invariably caused by an impact against the side of the head which produced shortening of the lateral axis of the skull. The fracture ran forward and downward in the majority of cases, running transversely through the floor of the middle fossa toward the sella turcica or the sphenoidal fissure. They often extended beyond the boundaries of the fossa to the anterior fossae or the middle fossa on the opposite side (figs. 4 and 5).

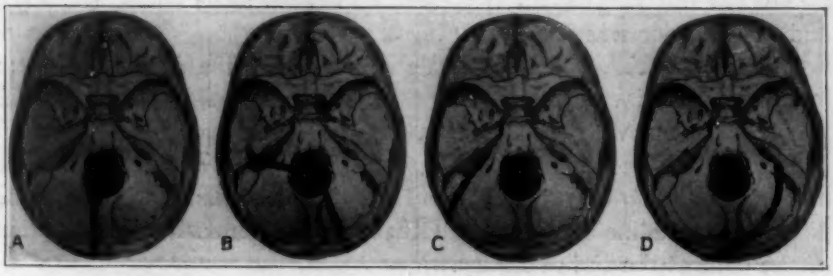

Fig. 2.—Types of posterior linear fractures. *A* represents linear fracture running into the foramen magnum; *B*, linear fracture crossing the foramen magnum and invading the petrous portion of the temporal bone on the opposite side; *C*, linear fracture running into the jugular foramen; *D*, linear fracture crossing posterior fossa to end in the jugular foramen on the opposite side.

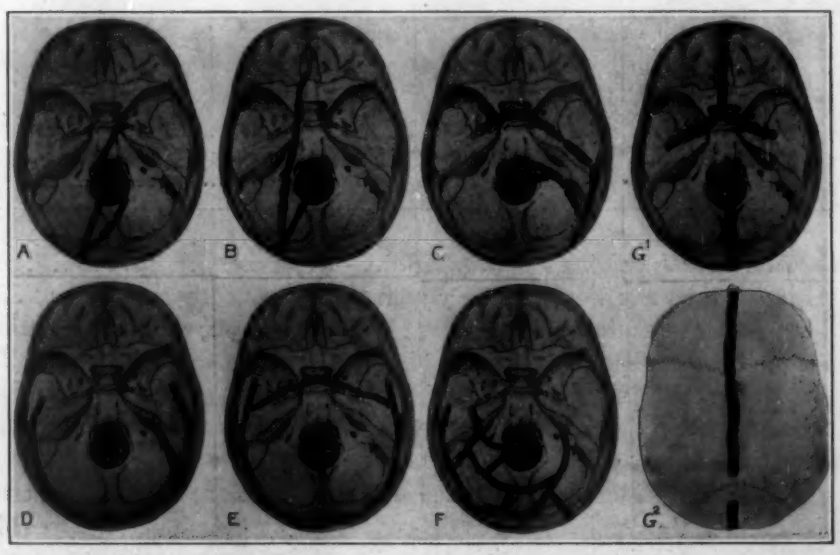

Fig. 3.—Types of posterior composite fractures. *A* represents angular type of posterior composite fracture; *B*, angular type of posterior composite fracture; *C*, dendritic type of posterior composite fracture; *D*, dendritic type of posterior composite fracture; *E*, extensive posterior composite fracture where the fracture lines joint at the sella turcica and isolate the posterior portion of the base from the rest of the skull; *F*, commiuted type of posterior composite fracture; G^1 and G^2, base and vault distribution of a posterior composite fracture which practically bisects the skull in the sagittal plane.

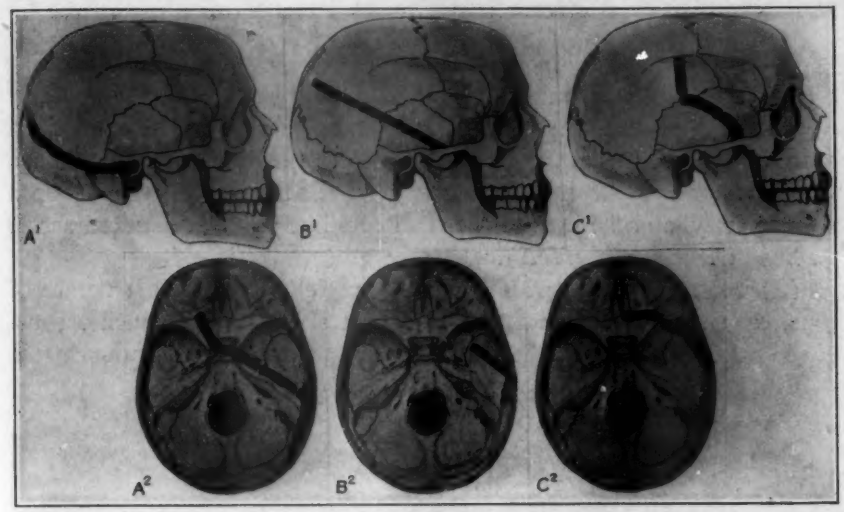

Fig. 4.—Types of lateral linear fracture. A^1 and A^2 represent vault and base distribution of a lateral linear fracture which involves the lambdoid suture, the roof of the middle ear and extends across the sella turcica to the anterior fossa of the opposite side; B^1 and B^2, the vault and base distribution of a lateral linear fracture involving the parietal bone and the middle fossa; C^1 and C^2, the vault and base distribution of a lateral linear fracture which involves the temporosphenoidal suture line, crosses the sphenoidal fissure and ends in the anterior fossa of the same side.

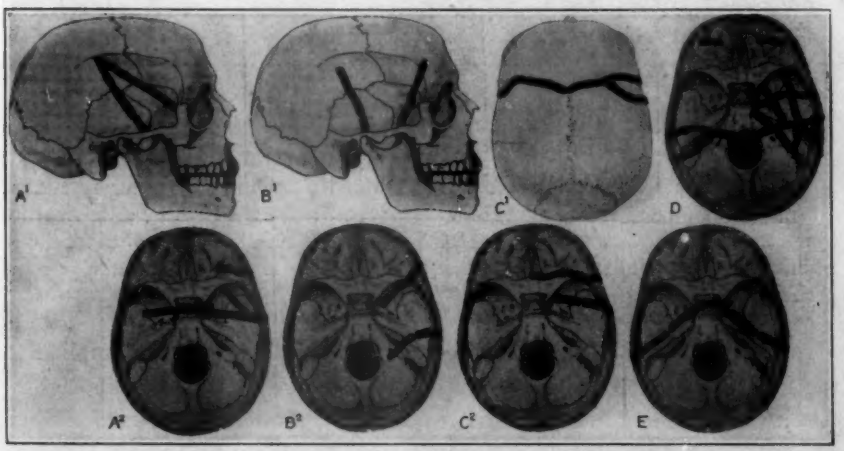

Fig. 5.—Types of lateral composite fracture. A^1 and A^2 represent the vault and base distribution of an angular type of lateral composite fracture; B^1 and B^2, the vault and base distribution of a lateral composite fracture composed of two approximately parallel lines; C^1 and C^2, the vault and base distribution of a lateral composite fracture which tends to divide the skull transversely in the region of the coronal suture; *D*, comminuted type of lateral composite fracture; *E*, lateral composite fracture caused by the crushing force of an automobile wheel which bisects the base of the skull.

Some peculiar types of lateral fracture were produced by a crushing force and not by a sudden impact. The causative agent was invariably an automobile. The wheel passed over the head, compressing the skull laterally and, as a rule, the base splint across the middle fossae, though other areas were involved as well. The fissures usually gaped open rather widely, and the base of the skull could be readily separated across the line of fracture (fig. 5 *E*).

(3) Anterior Location: The anterior region is a fragile structure of bone, easy to fracture, but apparently not subject to so much trauma as the other portions of the skull. Consequently, the fractures in the anterior region were comparatively few, only 12.5 per cent of the total

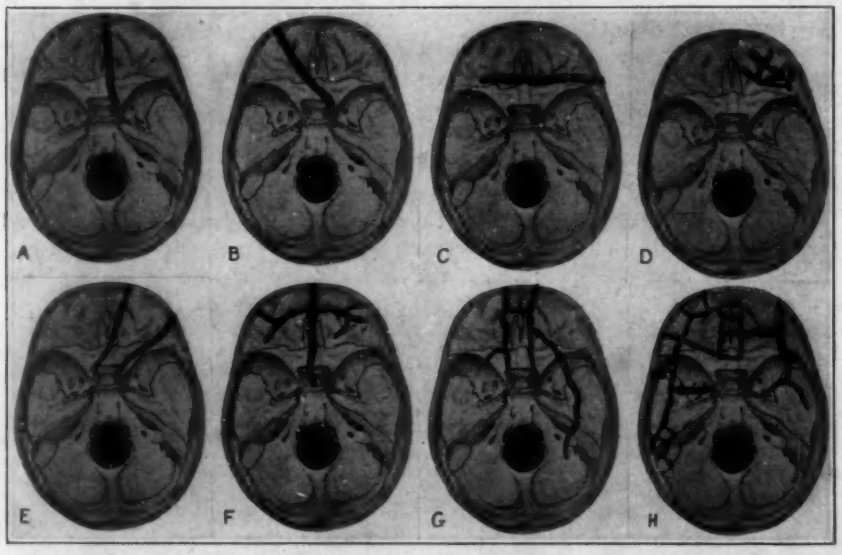

Fig. 6.—Types of anterior fracture. *A, B* and *C* are anterior linear fractures; *D*, small comminuted fracture of the anterior fossa; *E*, anterior composite fracture composed of two approximately parallel lines; *F*, anterior composite fracture; *G*, anterior composite fracture; *H*, extensively comminuted type of anterior composite fracture.

number. Most anterior fractures were the result of violence applied to the front part of the head, which resulted in a shortening of the sagittal diameter of the skull. The lines of fracture traveled backward toward the optic foramina and sometimes extended into other portions of the base. Occasionally the impact occurred in the lateral part of the frontal region, in which event a transverse fissure across both anterior fossae was produced (fig. 6).

The orbital plates were sometimes broken when some other part of the skull was struck. Any connection between the fracture arising near the point of impact and the orbital lesion often was not obvious. The

name of "contre coup fracture" has been given to such orbital cracks, but the term is meaningless. The orbital roof merely broke because it was especially vulnerable to strain and was unable to withstand the change of shape induced by the violence.

Fractures of the Vault.—Fractures of the vault were comparatively uncommon, forming only 6.7 per cent of the total number. Usually only those lesions were included in which the lines of fracture were confined mostly to the vault. Exceptions were made, however, in two cases in which the skull was extensively comminuted, both vault and base, due to an extreme grade of violence applied to the top of the head. Such injuries seemed to fit into the classification more logically here than elsewhere (fig. 7).

Of the different varieties, linear fractures were found in the parietal region. They were all short, and most of them ran downward and forward. One, however, ran downward and backward. They were, in all probability, produced by impact against the lateral part of the skull.

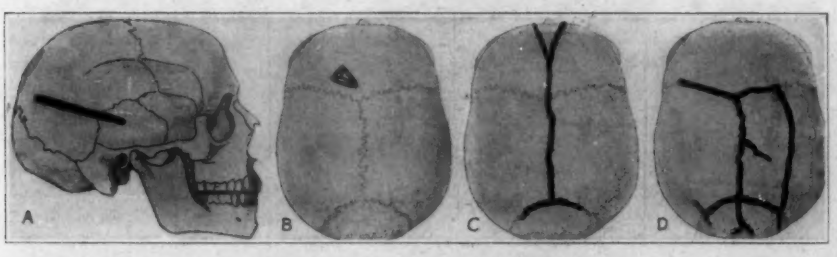

Fig. 7.—Types of vault fracture. *A* is a linear fracture of the vault; *B*, a depressed fracture of the vault; *C*, a composite fracture of the vault; *D*, a composite fracture of the vault.

Composite fractures generally were found on the vertex of the vault. Most of them were extensively comminuted, but a small number showed only a few branching lines.

Depressed fractures were a special type confined to the vault. They were caused by an object with a limited area of impact coming in contact with the skull; in most instances, this was a blunt instrument wielded with homicidal intent. A few were probably caused by falls against a projection. The appearance of the depressed fracture depended on the size and shape of the blunt instrument. The edge of a flatiron produced a triangular depressed fracture. A blackjack caused a roughly ovoid depression. In fact, the character of the depressed fracture not infrequently gave an important clue to the type of blunt instrument that was used (fig. 8).

In this connection, it is important to note that many fractures caused by blunt instruments were not classified as depressed fractures of the vault. The instrument was often large and the blow violent, so that the

fracture lines sometimes extended to other regions. It seemed more fitting to enter these lesions elsewhere in the classification.

Two freak examples of fracture occurred which were included, for the sake of convenience, among the composite fractures of the vault and base. The force which produced them, however, was unusual. The persons in question fell, striking on the vertex of the head, and both sustained fractures of the base, around and just anterior to the foramen magnum. There was evidence that the weight of the body, transmitted through the spinal column, forced the articular process of the occiput up through the base of the skull for a short distance.

Fig. 8.—Small depressed fracture in the squamous portion of the left temporal bone caused by the knuckle of a fist.

CAUSES OF DEATH

An attempt was made to ascertain the cause of death in each of the 507 cases so far as it could be determined from the postmortem examination. The clinical data were incomplete and, in most instances, gave only the length of time that the patient survived the injury. This information was too meager to allow an adequate study of the way in which the fatal complication developed during life. The end stages of the process, as disclosed at necropsy, were practically the only reliable facts obtainable, and on these the classification had to be based.

It was obvious that the intracranial conditions found at postmortem examination were not necessarily identical with the intracranial condi-

tions present in the living subject. As information about the latter was lacking, it was difficult in many instances to estimate the factors responsible for the fatal result. The cause of death, therefore, except in well marked cases, could not be divided into hard and fast categories, and the classification was principally valuable in that it offered a convenient outline for a review of the subject. The classification shown in table 3 was used:

Deaths from Cerebral Concussion.—Whenever the head is severely injured, a state of shock characterized by unconsciousness is immediately produced, which has been called cerebral concussion. All that can be said about this condition, as far as its etiology is concerned, is that the violence in some way jars the brain, and that the cerebral centers are depressed. It must be regarded solely as a clinical entity with a pathologic basis which our present methods of examination cannot demonstrate.

TABLE 3.—*Causes of Death*

Cerebral concussion	139
Exhaustion	14
Secondary pneumonia	27
Cerebral compression, lacerations of the brain and subdural hemorrhage	156
Cerebral compression and extradural hemorrhage	61
Meningitis and other septic conditions	48
Operation	4
Epilepsy	3
Other injuries	30
Natural causes	25

As a rule, the more extensive the fracture of the skull or the bigger the injury of the brain, the more profound is the degree of cerebral concussion. Fatal cases, however, occur without any fractures of the skull and without any visible evidence of brain injury. Evidently, there is not any necessary connection between the concussion and the actual physical lesions in the cranial organs.

The seriousness of the concussion depends on the severity of the violence and on the vitality of the person injured. Even in a healthy person, an extreme grade of violence will cause death in a few minutes. In one who is sick, senile, alcoholic or in other respects below par physically, a slight injury may be just as fatal. Others, however, rally against the shock and recover consciousness rapidly, although at times the ill effects of the condition may remain for several hours.

In the series of 507 cases, deaths from cerebral concussion occurred in 139. The majority of these patients died during the first hour after the injury, and none survived more than ten hours. The figures were as follows: deaths in a few minutes, 86; deaths in from one to four hours, 34; deaths in from five to ten hours, 11; unknown duration, 8.

The age incidence was not anything extraordinary and is recorded in table 4.

The casualties which produce the concussion were, in general, of the more severe types.

It was evident that the more severe fractures, as represented by the composite type, outnumbered the less extensive varieties in the ratio of over 3:1. Fractures in the posterior part of the skull were relatively numerous. Severe lacerations or bruises of the brain were also present in most cases, but they were entirely lacking in sixteen.

The variety of lateral composite fracture which was produced by the crushing force of an automobile wheel occurred in thirty-four cases. Most of the victims were children under 10 years of age. As a rule, this injury was rapidly fatal.

TABLE 4.—*Deaths Caused by Cerebral Concussion*

Years	No. of Cases	Years	No. of Cases
Age Incidence			
1 to 10	25	40 to 50	28
10 to 20	8	50 to 60	24
20 to 30	17	60 to 70	19
30 to 40	16	70 to 80	2
Casualty Incidence			
Automobile accidents	72	Knocked down in assault	2
Street car accidents	7	Struck by moving object	1
High falls	12	Struck by falling object	3
Low falls	14	Struck by blunt instruments	2
Falls down stairs	12	Unknown casualty	12
Thrown down in assault	2		

Different Types of Fracture	
Vault and base fractures:	
Posterior linear fractures	25
Posterior composite fractures	37
Lateral linear fractures	5
Lateral composite fractures	51
Anterior linear fractures	2
Anterior composite fractures	9
Vault fractures:	
Composite fractures	10

Alcohol was found by chemical examination in the brain and liver in forty of the 139 cases of cerebral concussion, or in a ratio of 28.6 per cent. Alcoholism and casualty have always been connected closely, and the action of the drug on the central nervous system of the injured person may exert a threefold influence on the trauma: First, the movements of an intoxicated man are incoordinated, and he is prone to blunder into accidents; second, the anesthetic action of the drug has a tendency to mask the symptoms of the injury and so render the diagnosis difficult; third, its depressing action on the vital nervous centers tends to emphasize the depressing effect of cerebral concussion, and death is likely to occur under a moderate degree of violence. Alcohol was also found in the viscera of skull fractures of persons who died of some cause other than concussion. However, owing to the fact that many of these patients lived so long that all the alcohol in the body had been excreted at the

time of death, the figures are not presented here. The patients whose accidents came under the category concussion lived only a few hours at the most and were the most suitable group for the consideration of the incidence of alcoholism in skull fractures.

Deaths from Exhaustion.—Fourteen persons with fracture of the skull lived from one to twenty days; at necropsy, an obvious cause of death could not be found. Many of these were elderly persons or people in a low state of vitality. These were classified as cases of death caused by exhaustion. The slow decline was considered the result of the concussion incurred at the time of the injury. The patients rallied against the shock to a certain extent, but the central nervous mechanism had somehow been injured by the jarring, and the vitality of the person was

TABLE 5.—*Deaths from Exhaustion*

Years	No. of Cases	Years	No. of Cases
Age Incidence			
1 to 10	1	60 to 70	4
30 to 40	2	70 to 80	3
40 to 50	2	80 to 90	1
50 to 60	1		
Casualty Incidence			
Automobile accidents	7	Thrown down, assault	1
Street car accidents	1	Hit by blunt instrument	1
Falls down stairs	3	Hit by falling object	1
Vault and Base Fractures			
Posterior linear fractures			3
Posterior composite fractures			1
Lateral linear fractures			3
Lateral composite fractures			6
Anterior composite fractures			1

not sufficient for him to throw off the depressing effects of the violence. In this group four persons lived from one to two days after being injured; two persons lived from two to three days; three persons, from four to six days; five persons, from nine to twenty days.

The composite fractures outnumbered the linear fractures in the proportion of 8 to 6. Among the lateral composite group was a crushing fracture caused by an automobile in a boy, aged 8, who lived for four and one-half days after the accident. Lesions of the brain were present in all instances, but in only one was the lesion of any significance. Lobular pneumonia was not found, nor was there any obvious reason for the patient's death besides the fractured skull.

In one instance, a contusion of the cerebral cortex contributed to the fatal termination. This was in a man, aged 40, who had been struck in the right parietal region with an iron bar. A depressed fracture with a linear extension in to the right middle fossa resulted from the blow, causing a broad bruise of the motor area of the right cerebral

cortex. The brain lesion produced numerous convulsive seizures, and death from exhaustion followed after a stormy course of six days.

Deaths from Terminal Lobular Pneumonia.—Persons with fractures of the skull who survive their injuries more than two days not infrequently develop lobular pneumonia as a terminal condition. This was shown at necropsy in fifty-nine patients out of the total of 507, but thirty-two of these died obviously of another complication, such as intracranial hemorrhage or suppurative meningitis. The other twenty-seven, however, did not show any other fatal lesions either in the head or in the rest of the body, except the terminal pneumonia.

Anatomically, the pneumonia was of the hypostatic lobular type, for the process was generally located in the posterior portions of the lungs. In the early stages it was often seen in the form of small, granular, gray-red areas of consolidation grouped around the small bronchioli. Later, these areas tended to coalesce, and a pneumonia similar to the lobar type was the result.

Two persons lived from from twelve to twenty-four hours after the fracture; five lived for from one to two days; nine, from two to four days; seven, from four to six days; three, from seven to nine days. An unknown person in a hospital lived eight hours.

Pneumonia usually developed after the second day, but some cases ran a slightly more rapid course. In the more acute types the question arose as to whether it was not possible that the lung condition might have antedated the fracture of the skull. This was not always easy to decide. If blood was inhaled, or the patient was exposed to cold, it was not unusual for pneumonia to develop rapidly. It was also common for many persons to have a "walking" pneumonia for several days, so that they could readily fracture the skull after the lung inflammation was developed fully.

The age incidence is shown in table 6.

The age incidence made it evident that the majority of the patients were middle aged and elderly persons. Their reaction to the trauma was similar to that described in fractures of the neck of the femur in the aged. The patients were never able to rally against the primary shock, but sank to the terminal hypostatic pneumonia.

The primary concussion with its accompanying state of unconsciousness undoubtedly played an important rôle in the development of the inflammation of the lung. There were, however, numerous traumatic intracranial lesions accompanied by hemorrhagic extravasations, which contributed something to the downward course. Twenty-three patients had a laceration of the brain of some kind, and the four who did not showed large hematomas either on the scalp or on the eyelids. Six patients had small extradural hemorrhages. None of these injuries in themselves would have proved fatal, though it is not unlikely that the

continual absorption of toxic products from the areas of blood extravasation had a depressing effect on the vitality of the patient.

Deaths caused by Cerebral Compression, Lacerations of the Brain and Subdural Hemorrhage.—(*a*) Pathologic Anatomy of Brain Injuries: Injuries of the brain are the most common complications produced by fractures of the skull. In the series of 507 patients, the brain was unharmed only forty-two times, while the remaining 465 patients had a cerebral lesion of some kind. The majority of these, however, did not produce any untoward effects, and it was estimated that only from 33 to 40 per cent played an important rôle in causing the death of the patient.

TABLE 6.—*Deaths from Terminal Lobular Pneumonia*

Age Incidence			
Years	No. of Cases	Years	No. of Cases
20 to 30	1	60 to 70	8
30 to 40	2	70 to 80	6
40 to 50	4	80 to 90	1
50 to 60	5		

Casualty List	
Automobile accidents	15
Street car accidents	2
Low falls	2
High falls	4
Unknown	4

Fracture Distribution	
Vault and base fractures:	
Posterior linear fractures	2
Posterior composite fractures	1
Lateral linear fractures	8
Lateral composite fractures	7
Anterior linear fractures	4
Anterior composite fractures	3
Vault fractures:	
Composite fractures	2

The injuries of the brain were mainly on the surface of that organ and resulted from the soft cerebral tissue coming in violent contact with the skull. All degrees of the lesion were present. A slight impact caused a localized subarachnoid hemorrhage by rupturing small vessels in the subarachnoid space. A greater violence bruised the cortex but left the arachnoid intact. Sometimes the force was severe enough to tear the arachnoid membrane and to lacerate the brain tissue superficially. At other times the tear was deeper, and considerable destruction of the gray matter was the result. In a few cases a vessel in the white matter near the surface was ruptured, and a blood cyst was found; some of these contained an ounce of blood clot; they usually opened into the subdural space, though sometimes they communicated with the lateral ventricles or formed closed cavities in the white or gray matter.

The torn vessels in recent lacerations bled into the subdural space in varying degrees. When the hemorrhage was slow, clots were formed

which adhered to the surface of the laceration and thus were localized in the injured area. A more rapid hemorrhage tended to fill the corresponding subdural space.

The subdural hemorrhages were either unilateral or bilateral. When unilateral, the blood was confined strictly to the subdural cavity surrounding the injured cerebral hemisphere. The hemorrhage never extended beyond the limits of this space, either to the opposite subdural space or below the tentorium. As the hemorrhage grew in size, the blood forced the brain over against the opposite vault of the skull, causing a noticeable flattening of the cerebral convolutions in this region. The convolutions on the side of the hemorrhage, however, were well marked and stood out prominently, although the arachnoid was pressed closely to the cortical surface. In those cases in which the skull was removed so that the underlying membranes were not torn, the dura was distended and tense from the pressure within.

The subdural hemorrhage was bilateral when lacerations occurred on both cerebral lobes. Either the two spaces contained approximately the same amount of blood or one side yielded a much greater amount than the other. The hemorrhages were not uniform in size, and there was not necessarily any relation between the nature of the laceration and the quantity of blood in the subdural space. With two lacerations of similar degree on either cerebral hemisphere, one might bleed a little, while the other might produce a profuse extravasation. Flattening of the cerebral cortex was not prominent in bilateral hemorrhages unless one side showed a large quantity of blood while the other contained only a small amount. However, when both subdural spaces were distended by the extravasation, the brain was pressed downward. Occasionally this produced flattening of the pons against the basilar process and a circular furrow of the inferior surface of the cerebellum by pressure against the rim of the foramen magnum.

Subdural hemorrhages below the tentorium were caused by cerebellar lacerations and were rather rare. Most of these were insignificant.

Subarachnoid hemorrhages were also found, and some of them were profuse. In a few instances, the space around the circle of Willis was distended by blood, which took its origin from some ruptured vessel in that network. These extravasations were spontaneous in most cases, usually were fatal and often were found without a history or evidence of injury of the head, but a few were undoubtedly traumatic. It was always difficult to make a distinction if a fracture was present.

Most subarachnoid hemorrhages of unquestionable traumatic origin were found on the upper portions of the frontal lobes. The blood was usually extravasated under the membrane from brain injuries near the tip of the lobe. The subarachnoid space was roomy on the cerebral vault but was closely applied to the convolutions at the base, so that the

blood spread profusely over the vertex of the brain and rarely spread down on the under surfaces. This lesion probably produced symptoms but in itself was not necessarily lethal.

In a few cases, the laceration opened into one of the ventricles of the brain, so that the cavity of the brain contained blood and blood clot, sometimes enough to cause an appreciable distention, such as would occur in internal hydrocephalus.

In some instances, small hemorrhages of traumatic origin were found in the pons and also in the basal nuclei underneath the floor of the lateral ventricles. These lesions were composed of multiple extravasations of blood varying in size from that of a pinhead to that of a pea, which sometimes tended to coalesce and form larger areas. Thus they conld be distinguished from spontaneous hemorrhages in similar locations, as the latter usually appeared as large solitary clots. At times, the distinction was difficult, for a spontaneous apoplectic stroke could

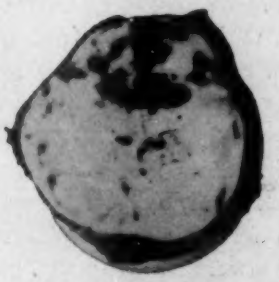

Fig. 9.—Cross-section of the pons showing small hemorrhages of traumatic origin.

occur at the same moment that the skull was fractured. As a rule, however, the traumatic pontile lesions were associated with severe fractures in the back part of the skull which in some way violently compressed the brain stem. Likewise, violent impacts near the external angular process of the frontal bone had some influence in producing the multiple lesions in the basal cerebral nuclei. Just how much of a factor these multiple hemorrhages were in causing the death of the patient is a question, because the degree of concussion was so severe that other symptoms were overshadowed. Some of the pontile hemorrhages, however, were large enough to cause distention of that area, and it was conceivable that this produced considerable trouble in itself (fig. 9).

Different stages in the healing of brain lacerations were often observed. Shortly after the injury, the lacerated area was bright red, but its color gradually changed to brownish red, then to brown and finally to a shrunken tan area known as "plaque jaune." The process was a gradual transformation of the blood pigment, the absorption of

necrotic débris and the development of a characteristic granulation tissue, with the final healing by scar tissue. The stages were analogous to the healing of wounds elsewhere in the body although, of course, not identical, as the brain had a characteristic cellular reaction which differed from any other tissue.

In some instances, adhesions between the brain and dura were found around the site of old lacerations. Granulation tissue, usually vascular, had grown into the injured area and had joined the two surfaces. Some of these adhesions were large and most often were found near the frontal and temporal regions in which they were always a potential seat of disturbance in ways which will be considered later.

Changes were also described in many subdural hemorrhages when they were seen long after the injury. A small hemorrhage might disappear and only a diffuse spotted, tan pigmentation on the inside of the dura would be left to indicate its presence. A somewhat larger extravasation was found in the later stages as a tan fibrinous membrane closely attached to the dura. More often, in persons who died from large extravasations, the clots varied in color from a wine red to a brownish red or cinnamon brown, and were more or less attached to the dura. Microscopic examination often demonstrated the fact that the dura showed a distinct inflammatory reaction against the extravasated blood.

(*b*) Relation of Brain Lacerations to Fractures of the Skull: Brain lacerations were classified according to their method of production into (1) direct lacerations and (2) contre-coup lacerations.

(1) Direct lacerations were so designated because the surface of the brain was torn directly by the inward depression of the sharp fracture edges which often inflicted a corresponding tear on the adjacent dura. The form and position of the laceration, therefore, depended on the variety of the fracture.

Linear fractures produced furrows of different dimensions which marked the course of the fissure in the skull on the brain. Important lesions of this type were most often found on the lateral aspect of the parietal and temporal lobes, for the broad and flat expanse of skull which covers this area is easily depressed by force. The cerebral laceration was occasionally severe enough to produce a fatal subdural hemorrhage. Longitudinal linear fractures of the posterior fossa often furrowed the under and outer surfaces of the cerebellar lobes. In a similar fashion, linear fractures of the anterior fossae occasionally caused a linear laceration of the orbital convolutions of the frontal lobes. These, however, as a rule, were not productive of any alarming complications.

Composite and depressed fractures also produced characteristic lacerations in the form of fossae and pits of various sizes and shapes. While they occurred almost anywhere on the surface of the brain, the

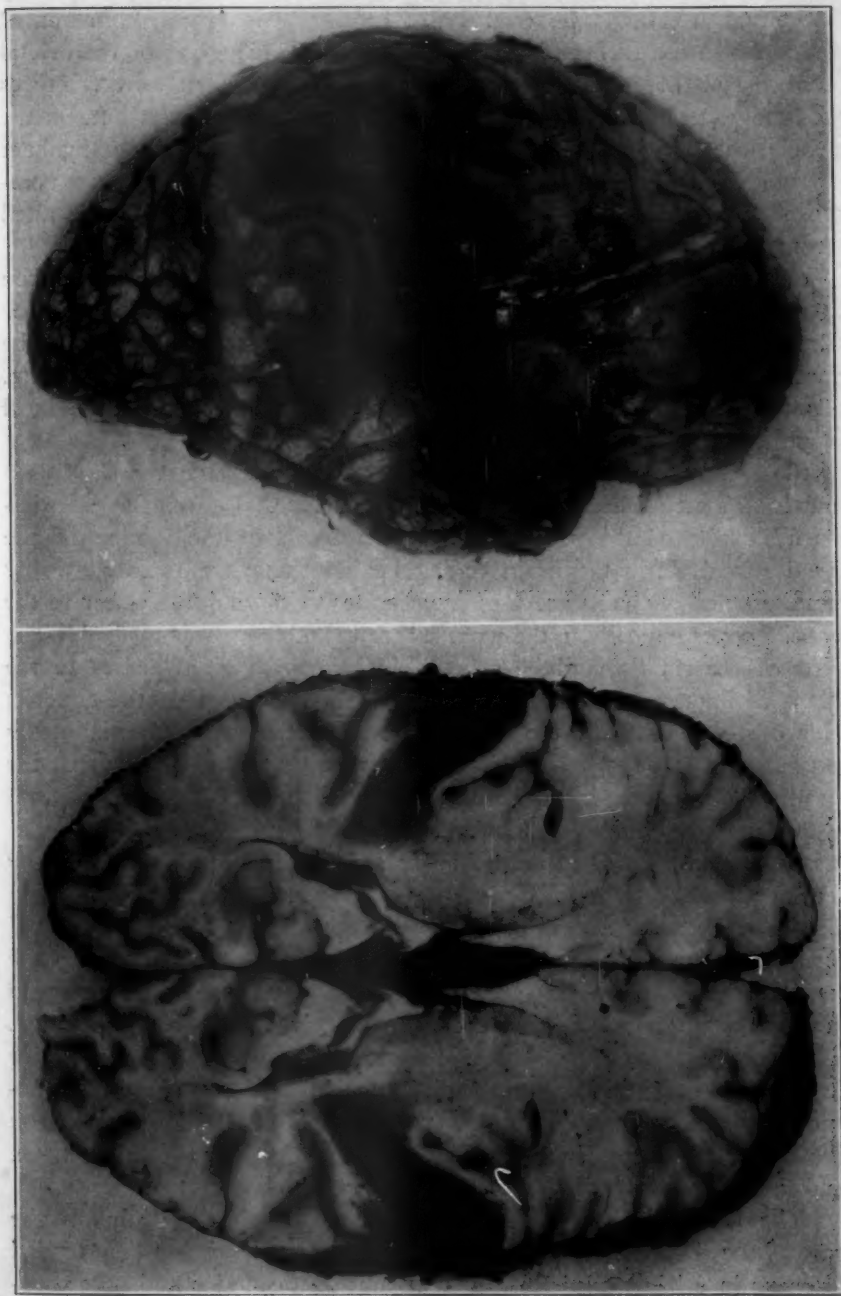

Fig. 10.—Direct laceration of the outer surface of the right cerebral hemisphere caused by a lateral composite fracture of the right parietal and temporal regions. The depth of the laceration is shown in the section of the brain.

external parietal and temporal areas suffered most and showed some deep lacerations of this type (fig. 10). In a few instances, comminuted fractures in the frontal region caused extensive mangling of the orbital convolutions of the frontal lobes. A few posterior composite fractures at times lacerated the lateral occipital areas rather severely, but, on the whole, this was rare, as the sharply curved occiput is not easily bent by violence. In one case, the cerebellum was severely torn by a composite fracture in the posterior fossa, so extensively that death resulted from the subsequent intracranial hemorrhage.

(2) Lacerations of the brain were designated as contre-coup when it was evident that they were produced by a force which fractured the skull at the point of impact and at the same time caused the brain to be lacerated by violent contact with the opposite side of the cranium. Contre-coup lesions, therefore, were found on the side of the head opposite the one on which the fracture took its origin.

The contre-coup laceration generally occurred when the skull as a moving object struck violently against a surface. LeCount and Apfelbach [1] explained this mechanism by the fact that the motion of the head would cause the brain to lag behind the more rapidly moving cranium, and at the moment of impact the brain would be closer to the cranial bones opposite to the place where the violence was applied. The axis of the skull would be shortened abruptly at right angles to the plane of impact, and this would lacerate the brain against the opposite side of the skull. Most of the contre-coup lesions were undoubtedly produced in this fashion, and the essential factor seemed to be that the head was in motion or free to move at the time of casualty.

Contre-coup lesions never occurred when the skull was held motionless at the time of fracture. This point was also duly emphasized by LeCount and Apfelbach. It was well illustrated in the forty fractures produced by an automobile wheel crushing the head of the deceased against the roadway—cases in which motion was not imparted to the head by the violence. Sometimes a few direct lacerations of the brain resulted, but no unmistakable contre-coup laceration was identified. The same point was also shown in a man who was struck with a table leg as he lay with his head on a pillow. There was an extensive fracture and a direct laceration at the point of impact, but a laceration on the opposite side was not present. The head was not free to move, and the contre-coup mechanism could not work.

The location of contre-coup lacerations depended mainly on the part of the skull that was fractured, and it was noticed that they tended to occur in fairly well defined areas, namely, the under and outer sur-

1. LeCount, E. R., and Apfelbach, C. W.: Pathologic Anatomy of Traumatic Fractures of Cranial Bones and Concomitant Brain Injuries, J. A. M. A. **74**:501 (Feb. 21) 1920.

faces of the frontal and temporosphenoidal lobes. The brain rested directly on the dura in these areas, and, because of its close proximity to the skull, was easily lacerated by the contre-coup violence accompanying posterior or lateral fractures. The brain injury was often severe and was attended by fatal intracranial hemorrhages.

When the impact was in the posterior region of the skull, the under surfaces of the two frontal and two temporosphenoidal lobes were usually lacerated, sometimes all four, sometimes three, two or even one (fig. 11). There was no regularity as the extent of the involvement.

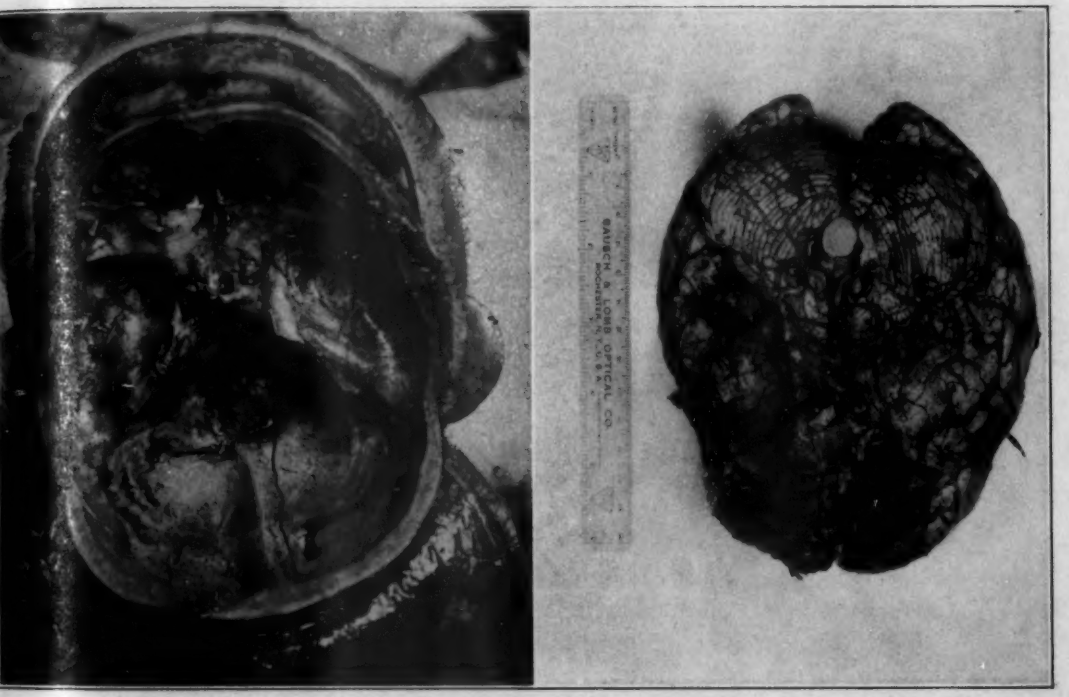

Fig. 11.—Posterior linear fracture of the base of the skull accompanied by broad shallow contre-coup lacerations of the under surface of the left temporal lobe and both frontal lobes.

At times, the anterior tips of the lobes would suffer instead. In other instances, the lateral surfaces on one side would be injured, especially if the point of impact in the occiput was a little to one side of the median line.

A lateral impact generally produced a contre-coup laceration on the opposite temporosphenoidal lobe, either on the lateral aspect or on the under surface of the lobe. Not infrequently, this was combined with a direct brain laceration on the side of the fracture.

Occasionally a lateral fracture was associated with a contre-coup bruise on the vertex of the brain in the midline near the coronal suture. This lesion was rarely dangerous in itself, but not infrequently accompanied tearing of the tributaries of the superior longitudinal sinus, which will be discussed later. Another effect of lateral violence was the presence of small pin-point hemorrhages in the corpus callosum, probably caused by impact against the falx cerebri. This also was of minor importance.

Force applied to the anterior part of the skull rarely produced a contré-coup lesion; when such a lesion was produced, it was only of slight grade. The occipital lobes and the cerebellum occasionally showed slight contusions, but none of these were significant.

TABLE 7.—*Deaths Caused by Cerebral Compression, Lacerations of the Brain and Subdural Hemorrhage*

Years	No. of Cases	Years	No. of Cases
Age Incidence			
1 to 10	3	40 to 50	40
10 to 20	1	50 to 60	32
20 to 30	14	60 to 70	16
30 to 40	19	70 to 80	7
Duration of Clinical Course			
1 to 6 hours	32	4 to 6 days	5
7 to 12 hours	27	6 to 8 days	7
13 to 24 hours	21	8 to 10 days	5
1 to 2 days	10	14 days	2
2 to 4 days	14	Unknown	9
Casualty List			
Automobile accidents	31	Falls down stairs	23
Street car accidents	7	Knocked down, assault	5
Bicycle accidents	1	Hit by moving object	2
High falls	9	Hit by blunt instrument	3
Low falls	14	Unknown casualty	37

Impact directly on the vertex of the skull at times resulted in the contre-coup lacerations of the orbital convolutions of the frontal lobes or on the under surface of the temporal lobes.

Sometimes recent brain lacerations were found at necropsy when fracture of the skull was not present. They were invariably produced by contre-coup violence, usually resulting from a fall, and they were occasionally followed by a fatal subdural hemorrhage. The point of impact could be located by the scalp wound. Cases of this kind, however, were not included in this series and are mentioned only to indicate that brain lacerations were not invariably dependent on fractures of the skull.

(*c*) Types of Subdural Hemorrhages and Lacerations of the Brain Causing Death by Cerebral Compression: The deaths in this category were due to cerebral compression caused by (1) blood in the subdural space or (2) laceration of the brain. One hundred and thirty-two deaths were caused by subdural hemorrhages and twenty-four by lacerations of the brain.

(1) A case was listed as subdural hemorrhage whenever it was apparent that there was enough hemorrhage in the subdural space to produce intracranial pressure.

The majority of patients died within twenty-four hours, but others survived for several days.

The location of the fracture and the cause and extent of the subdural hemorrhage are shown in table 8. The subdural hemorrhages are classified as unilateral, in which the hemorrhage was in one subdural space, and bilateral, when both subdural spaces contained blood. The words direct and contre-coup indicate whether the source of the

TABLE 8.—*Fractures and Intracranial Lesions Causing Subdural Hemorrhage*

Origin of the Subdural Hemorrhage	Type of Fracture: Posterior Linear	Posterior Composite	Lateral Linear	Lateral Composite	Anterior Linear	Anterior Composite	Vault Linear	Vault Composite		
Unilateral Subdural Hemorrhages:										
Contre-coup lacerations of brain	20	18	20	9	0	0	0	0	67	
Direct lacerations of brain	0	3	6	8	0	0	1	1	19	
Direct and contre-coup lacerations of brain, combined	0	0	0	1	0	0	0	0	1	
Tearing of dura and middle meningeal artery	0	0	0	1	0	0	0	0	1	
Injury of old adhesion of brain to dura	0	0	0	0	1	0	0	0	1	
Torn vein, unilateral	0	0	0	0	0	1	0	0	1	
										90
Bilateral Subdural Hemorrhages:										
Contre-coup lacerations of brain	12	6	0	0	0	0	0	0	18	
Direct lacerations of brain	0	0	0	0	0	2	0	0	2	
Direct and contre-coup lacerations of brain, combined	1	0	6	10	0	0	3	0	20	
Contre-coup lacerations of brain and torn dural vein	0	0	1	0	0	0	0	0	1	
Torn venous tributaries of dural sinus	0	0	0	0	0	1	0	0	1	
										42
	33	27	33	29	1	4	4	1	132	

hemorrhage was from direct or contre-coup lacerations, and, whenever the source was from some other lesion, that point is sufficiently designated. It was found in all categories that right and left lesions were equally balanced; consequently, the point is not shown on the table.

The casualty and age tables did not contain anything significant.

An analysis of the fracture sites and the cause of the subdural extravasation, however, brought out some interesting points. The fractures occurred most often on the posterior and lateral portions of the skull, with comparatively few involving the anterior and vault regions. The hemorrhages were caused by contre-coup lacerations in eighty-five cases, by direct lacerations in twenty-one cases and by contre-coup and direct lacerations combined in twenty-one cases. The other intracranial injuries which caused this kind of bleeding were few in number and will be considered later.

The posterior fractures were most likely to result in contre-coup lacerations. In only three were serious direct lacerations alone present, and in these cases the lateral occipital regions were injured by an extensive composite fracture. In one case, a direct laceration and a contre-coup laceration on opposite sides caused a bilateral hemorrhage.

Lateral fractures caused forty-five unilateral subdural hemorrhages, of which twenty-nine were caused by contre-coup lacerations, fourteen by direct lacerations, one by a direct and contre-coup laceration in the same cerebral hemisphere and one by the tearing of the dura and a branch of the middle meningeal artery. Bilateral subdural hemorrhages occurred in seventeen instances, of which sixteen were the result of a direct laceration on the side of the fracture combined with a contre-coup laceration on the opposite side. The other case was the result of the tearing of the small tributary veins of the longitudinal sinus near the coronal suture, combined with a contre-coup laceration.

In one instance, the dura and a branch of the middle meningeal artery were torn by a fragment of bone, and profuse hemorrhage into the subdural space resulted. This was unusual, as most tears of the middle meningeal artery bled extradurally. However, the firm attachment of the dura to the cranium prevented an extradural extravasation, and the severe comminuted fracture in the temporal region caused the bleeding artery to point through the dural tear into the subdural space. The blood spurted upward and was found as a large compact clot, 220 Gm. in weight, compressing the parietal convolutions inward.

The vault fractures were all located laterally and caused unilateral subdural hemorrhages in two instances from direct lacerations and bilateral subdural hemorrhages in three instances from direct and contre-coup lacerations.

Fractures in the anterior region caused unilateral subdural hemorrhages in two instances, one from the tearing apart of old adhesions of the brain to the dura by the recent violence, and the other from a complete tear of a vein which connected the temporal lobe of the brain with the dura of the middle fossa. In both these instances the violence was applied just to the right of the midfrontal region in such a way that the skull was shortened in the diagonal diameter from front to back and from right to left. The brain in the left temporal region was forced to slide backward along the inside of the skull, and this produced the tearing of the aforementioned vessels.

Anterior fractures caused bilateral subdural hemorrhages in three instances. In two, a comminuted fracture of both orbital plates extensively lacerated the adjacent portions of the frontal lobes and produced the bleeding. In the other case, the force struck the lateral portion of the right frontal region, shattered the right orbit and imparted a violent lateral oscillation to the brain. The tributary veins which run

from the brain to the superior longitudinal sinus were torn on both sides near the coronal suture and bled profusely into the subdural spaces. These same tributary veins are not infrequently ruptured during a boxing match by a heavy blow applied to the side of the head, which, however, does not necessarily cause a skull fracture. The essential factor in the production of the venous rupture is the oscillation of the brain from side to side in such a manner that the venous tributaries near the vertex are under special stress.

In thirty-six cases of subdural hemorrhage, the extravasated blood was carefully detached from the dura and brain lacerations and weighed. When the hemorrhage was bilateral, the blood in both subdural spaces was combined, and the weight of this was taken.

The average weight of the subdural hemorrhage was 61.1 Gm.

When the clinical course was short, the clots were usually smaller, but patients who died within six hours often showed some rather large hemorrhages, one unilateral extravasation reaching the size of 110 Gm.

TABLE 9.—*Weight of the Clots in Subdural Hemorrhages*

Gm.	No. of Cases	Gm.	No. of Cases
15	1	70	3
20	1	80	5
25	3	90	1
30	4	100	2
40	2	110	1
50	7	130	1
60	4	220	1

The interesting feature was that the small subdural hemorrhages produced just as acute evidences of pressure as the large ones. Of course, the subdural spaces were of different capacities, but the variation in capacity, as represented by the weights of the subdural clots, was much greater than any normal variation. The intracranial organs, however, were not in a normal state after a severe skull fracture, and some conditions of the brain were often present that probably reduced the capacity of the subdural spaces. This is treated in more detail subsequently.

2. Twenty-four fractures of the skull showed severe brain lacerations which resulted in cerebral compression without causing any subdural hemorrhage worthy of note.

The lacerations of the brain were extensive, but they differed in their anatomic characteristics. Some were shallow, showing a depth about the thickness of the cerebral cortex, but covered a wide area, often as much as from 6 to 8 square inches. In all probability, they caused the intracranial tension because the traumatized brain tissue and adherent blood clot gave rise to a swelling of the brain substance around the site of the laceration from local circulatory disturbances. Several cases lasted for from two to five days, and an uninjured zone of cerebral

substance immediately adjacent to the broad surface lesion was swollen, translucent and obviously edematous. This swelling was sufficient to produce a definite intracranial tension.

Some lacerations, however, extended into the brain substance, forming large cavities near the surface, from 1 to 2.5 inches in diameter, which contained 20 to 30 Gm. of blood clot (fig. 12). The cause of the cerebral compression was obvious. In four instances, the cavity extended into the ventricles of the brain and filled them with blood clot.

TABLE 10.—*Cases of Brain Lacerations Which Caused Cerebral Compression*

Years	No. of Cases	Years	No. of Cases
Age Incidence			
1 to 10	2	40 to 50	7
10 to 20	1	50 to 60	5
20 to 30	1	60 to 70	2
30 to 40	6		
Casualty Incidence			
Automobile accidents	12	Falls down stairs	2
Low falls	2	Assault by blunt instrument	2
High falls	1	Unknown	5
Duration of Clinical Course			
1 to 6 hours	3	4 days	1
7 to 12 hours	8	5 days	2
13 to 24 hours	8	Unknown	1
2 days	1		

TABLE 11.—*Location and Variety of Brain Lacerations Causing Cerebral Compression*

Types of Lacerations	Types of Fracture				
	Posterior Linear	Posterior Composite	Lateral Composite	Vault Composite	
Unilateral contre-coup lacerations	2	3	1	..	6
Bilateral contre-coup lacerations	5	4	1	1	11
Direct laceration, cerebrum	..	..	3	..	3
Direct laceration, cerebellum	..	1	..	..	1
Direct and contre-coup lacerations	..	..	2	..	2
Direct and contre-coup laceration, unilateral	..	1	..	..	1
	7	9	7	1	24

Intraventricular hemorrhages: contre-coup lacerations of cerebrum, 3; direct laceration of cerebellum, 1.

Death usually occurred soon—within twenty-four hours. One patient, however, survived for four days. Three lacerations opened into the lateral ventricles, while the fourth was caused by a laceration of the cerebellum which bled into the fourth ventricle.

(*d*) Conditions of the Brain in Cerebral Compression: The first few hours immediately following a severe fracture often produced clinical signs of increased intracranial pressure, and cases which came to necropsy in this period showed anatomic evidence of this condition in flattened convolutions, engorged cerebral blood vessels and other

signs. The cause for this was not always evident, as the actual hemorrhage inside the skull did not seem sufficient to produce as much disturbance as was noted.

Cases which came to necropsy in from twelve to twenty-four hours after injury showed a distinct swelling of the brain, the arachnoid was under evident tension and slight unilateral hemorrhage produced marked flattening of the convolutions on the opposite side. On section, the brain tissue seemed a trifle moist, and the condition appeared analogous to that which Apfelbach [2] described as "edema of the brain." No case in this series, however, presented the degree of swelling that Apfelbach mentioned, and there was no example of death arising solely from

Fig. 12.—Sagittal section of the right cerebral hemisphere showing huge cavernous lacerations of the frontal and temporal lobes caused by contre-coup violence.

this condition. Any hint concerning its origin could not be found in the material, and the explanations so far advanced in the literature were not considered satisfactory in all respects. The fact can only be noted that the swelling was a real result of the trauma and helped to produce an increase in intracranial pressure.

The viscera in the trunk in cases of cerebral compression showed most noteworthy changes in the lungs. When the course was rapid and failure of the circulatory apparatus supervened, the lungs were the seat of marked congestion and edema, usually in the posterior portion. They were generally heavy and dripped blood profusely on section.

2. Apfelbach, C. W.: Studies in Traumatic Fractures of the Cranial Bones, Arch. Surg. 4:434 (March) 1922

Subdural hemorrhages, however, that lasted several days often showed hypostatic bronchopneumonia of the type already described.

Deaths Caused by Cerebral Compression and Epidural Hemorrhage.—In the series of 507 cases, epidural or extradural hemorrhage was described in 106. Forty-five of these blood clots were small and were not considered important. The remaining sixty-one, however, were large enough to prove fatal through cerebral compression.

All epidural hemorrhages in fractures of the skull are caused by the sharp edges of the broken bone lacerating a vessel on the outside of the dura. The torn vessel bleeds in between the dura and the skull and pushes the dura inward against the adjacent portion of the brain,

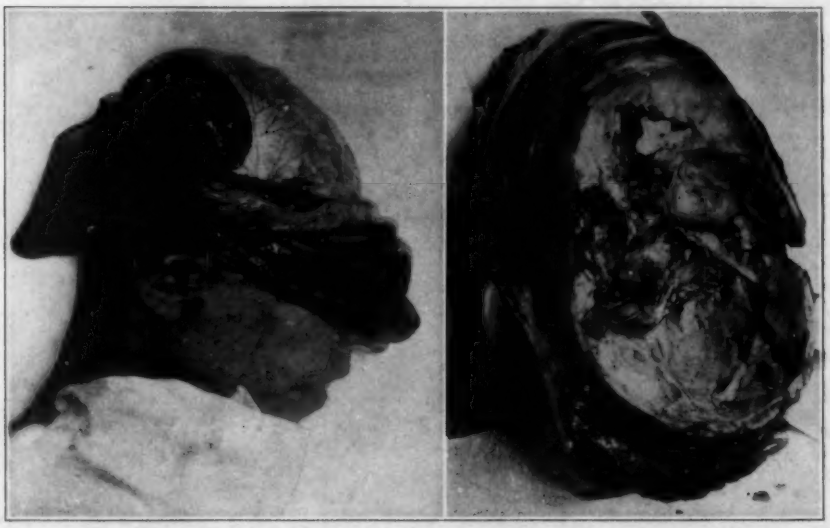

Fig. 13.—Middle meningeal hemorrhage in the right posterior parietal region. The lateral composite fracture and part of the craniotomy opening are also shown on the right side of the skull.

causing compression of that area. The bleeding occurs only where the line of fracture extends and is never found elsewhere. The vessels most often lacerated are the branches of the middle meningeal artery. In rare instances, the lateral venous sinus and the anterior meningeal artery are involved.

The middle meningeal artery is in a good position to be lacerated, as it is placed on the outside of the dura and lies in grooves on the inside of the skull. It enters the skull through the foramen spinosum in the middle fossa and runs laterally a short distance before it divides into anterior and posterior branches, which spread in a dentritic fashion over the temporal and parietal regions of the vault. It is, therefore, easily reached by lateral fractures of all kinds.

In this series, fractures in the lateral parts of the skull numbered 233, and epidural clots from the middle meningeal artery of any size numbered fifty-six, so that only a small proportion of fractures resulted in serious laceration of the artery. A reason for this is suggested by the changing relationship between the skull and dura at different periods of life.

In childhood, the bony grooves for the middle meningeal artery are shallow, and the dura is easily separated from the skull. Accordingly, the artery escapes injury except in rare instances. As the person grows older, the dura becomes more and more adherent, and the grooves deepen, so that in old people there is a thick adherent dura and pronounced grooves. Extradural clots are rather infrequent at this age, as

TABLE 12.—*Cases of Cerebral Compression and Epidural Hemorrhages*

Years	No. of Cases	Years	No. of Cases
Age Incidence			
*10 to 20	2	50 to 60	7
20 to 30	13	60 to 70	2
30 to 40	18	70 to 80	2
40 to 50	17		
Casualty Incidence			
Automobile accidents	5	Knocked down in assault	3
High falls	9	Struck by blunt instrument	5
Low falls	10	Hit by flying object	1
Falls down stairs	7	Unknown	21
Duration of Clinical Course			
1 to 6 hours	9	4 days	1
7 to 12 hours	13	6 days	2
13 to 24 hours	11	8 days	2
1 to 2 days	2	Unknown	17
2 to 3 days	4		

* Since this paper was written, the author has seen a case of a child, aged 8, who died from an epidural hemorrhage from the anterior branch of the middle meningeal artery. The cause was a comminuted lateral fracture. The condition in children is rare.

the adherent dura prevents excessive hemorrhage. In young adult life and middle age, however, the bony grooves are not so deep, and the dura is adherent just enough to allow laceration of the artery, but not to the extent that it prevents its separation from the bone by the violence. Most extradural hemorrhages are found at these ages, as shown in table 12.

The decade from 30 to 40 years contained the greatest number of cases.

One of the cases, that of a person aged 70 years, was noteworthy in that the skull was the seat of a pronounced osteitis deformans. The bone was thick but soft and pliable, while the dura was not firmly adherent to the skull. Conditions were therefore favorable for the epidural clot which developed.

The duration of the clinical course was rather variable, over one half of the patients dying inside of twenty-four hours. In other instances,

the patients lingered a few days. A large proportion were found dead, and it was impossible to discover not only how long they lived after the injury, but also what the casualty really was.

Many persons with epidural hemorrhages, especially those found dead or unconscious, showed extraordinarily thin skulls. They were, apparently, fractured by a comparatively slight violence, so that the primary concussion was of short duration or entirely absent. It was not unusual for these persons to wander away from the scene of the accident and to be overcome by rapidly developing cerebral compression some hours later. If found alive, they were either moribund or stuporous, which suggested alcoholic intoxication, and the serious nature of their condition was not recognized. The difficulties of diagnosis not infrequently were increased by the entire absence of any external injury to the head and also by the fact that the deceased was alcoholic. It was probable that the majority of these fractures were caused by falls.

TABLE 13.—*Epidural Hemorrhages and Their Source*

Vessels Ruptured by Fracture	Fractures, Types and Location								
	Posterior Linear	Posterior Composite	Lateral Linear	Lateral Composite	Anterior Fissure	Anterior Composite	Vault Linear	Vault Depressed	
Middle meningeal artery, posterior branch	..	4	11	6	..	..	3	1	25
Middle meningeal artery, anterior branch	..	1	5	3	..	..	1	2	12
Middle meningeal artery, main stem	..	..	2	..	..	2	..	..	4
Middle meningeal artery, branch involved not located	..	..	9	4	1	..	1	..	15
Anterior meningeal artery	..	..	..	..	1	..	..	..	1
Lateral venous sinus	3	1	..	..	..	..	..	..	4
	3	6	27	13	2	2	5	3	61

The form and location of the hemorrhage varied with the site of the fracture and the vessel that sustained the rupture.

The right and left sides were equally involved and were not indicated in the table.

Lateral fractures were responsible for forty epidural hemorrhages, almost two thirds of the total number, and the middle meningeal artery was the only vessel ruptured. Other fractures occasionally lacerated this vessel, but only when a fracture line extended to the parietal region or the middle fossa. In this connection, it was worthy of note that epidural hemorrhages did not result from the transverse crushing fractures of the skull which have already been described.

The total number of middle meningeal hemorrhages was fifty-six. Of these, twenty-five were identified as arising from the posterior branch, twelve from the anterior branch, four from the main stem in the middle fossa, and fifteen could not be located with certainty.

The only way to determine the origin of the hemorrhage at necropsy was by a consideration of the fracture lines and the position of the clot, as the tear of the artery could not always be found. In the fifteen undetermined cases, it was not possible to ascertain the site of the rupture even in this way, for the observations were not definite enough.

The middle meningeal hemorrhages occurred on the vault in most instances; only a few were found at the base. Torn posterior branches of the artery generally formed blood clots in the middle and posterior portions of the parietal region. Ruptures of the anterior branches bled outside the dura in the anterior portion of the lateral surface of the vault. Most hemorrhages of the vault were unilateral and showed a circular and ovoid outline, with a sharp rim and a thick center, so that they could be designated as disk, diskus or platter shape. If the artery was lacerated near the base, a small tongue of blood clot occasionally ran from the point of laceration upward to the disk-shaped hemorrhage above. In one instance, the line of fracture extended over the posterior parietal regions from one side to the other and ruptured the posterior branches on each side of the skull, forming two discrete, symmetrically placed, bilateral epidural hemorrhages of equal size.

In some cases, rupture of the anterior branches or the common stem caused an extradural hemorrhage at the base of the skull. The blood clot then developed a globular or egg-shaped form and pressed the under surface of the frontal and temporosphenoidal lobes upward.

In one instance, an anterior fissure fracture lacerated the anterior meningeal artery as it emerged from its foramen, forming a globular hemorrhage which dented the anterior tip of the frontal lobe.

Ruptures of the lateral venous sinus were the results of fracture in the posterior regions, which tore the posterior wall of the sinus. The hemorrhage took an ovoid form and pressed in the cerebellum and occipital lobes. These were especialy fatal, as this region of the brain was vulnerable to pressure.

The characteristics of the epidural hemorrhage varied with the size of the clot and the length of time that the patient lived after injury. Hemorrhages of recent origin at necropsy showed friable blood clots, resembling currant jelly, which were easily detached from the dura. Sometimes, when there was a large amount of extravasated blood, about 1 inch thick through its center, it was possible to find separation of the blood clots into layers such as occurs in postmortem clotting. Hemorrhages of longer duration were firm, compact, rusty red and adherent to the dura.

The blood between the dura and bone had an irritant action on the bone, but unfortunately this could not be studied satisfactorily in the material at hand. In the cases that came to necropsy, observations which could be positively identified as the remains of an old extradural hemor-

rhage of any size were not encountered, although occasionally peculiar, milky white, eburnated, roughened areas on the inside of the skull were found, to which the dura was firmly attached. The same observations were not infrequent around the edges of old decompression windows and occasionally in the region of old healed fractures. It was not improbable that these milky white areas were the remains of small epidural blood films which were frequently noted around fractures, and which were caused by the tearing of multiple small communicating vessels when the dura was separated from the bone during the violence.

The epidural clot was detached from the dura and carefully weighed in twenty-five cases. The weights are tabulated in table 14.

The average weight of the epidural hemorrhage was 122 Gm., which is about twice as large as the weight of the average subdural hemorrhage, which was 61 Gm.

There was not any essential correspondence between the weight of the blood clot and the duration of the clinical course, as small and large

TABLE 14.—*Weight of the Epidural Hemorrhage*

Gm.	No. of Cases	Gm.	No. of Cases
40	1	120	1
50	2	130	2
60	1	140	1
70	0	160	1
80	2	170	3
90	2	190	1
100	3	200	1
110	3	300	1

hemorrhages were found at all periods. There likewise was not any difference in size between the blood clots that came from the anterior or posterior branches of the middle meningeal artery. However, epidural hemorrhages from the lateral venous sinus were generally smaller than the middle meningeal artery hemorrhages, for the two sinus hemorrhages measured were only 50 and 60 Gm., respectively.

Other lesions were occasionally found in deaths from epidural hemorrhages. Lacerations of the brain were found in fifty-six cases but gave rise to subdural extravasations in only sixteen cases, of which thirteen were caused by contre-coup lacerations and three to direct lacerations. None of them were severe enough to cause a fatal cerebral compression in themselves. In five cases, brain injury of any kind was not visible.

The condition of the lungs was similar to that found in the cases of subdural hemorrhages. Death from cerebral compression occurring soon after an accident was accompanied by marked hypostatic congestion and edema of the lungs, while a longer clinical course sometimes showed a terminal lobular pneumonia.

Deaths Caused by Acute Suppurative Meningitis and Other Septic Infections Caused by the Fracture of the Skull.—In the series of 507 cases, forty-eight patients died of acute suppurative leptomeningitis or some type of general septic infection. In every case the source of the fatal infection was obviously the fracture of the skull. A tabulation of the age, casualty and duration of clinical course brought out many interesting points.

Meningitis: The table of casualties did not give any significant information. The age categories showed, however, that, while the adult decades were well represented, the proportion of children below 10 years of age was rather large. The clinical course was invariably of several days' duration, and not infrequently the patient lingered for several weeks.

The meningitis could be traced either to one of the bony sinuses of the skull or to infection which entered by means of a compound fracture. The probable source of the infection was tabulated in connection with the variety of the fracture.

Seven posterior fractures caused meningitis, and in every case they sent a branch into the mastoid and middle ear areas, in which the infection originated. Six of these bony sinuses showed a frank purulent exudate.

There were seventeen lateral fractures, and in every case the fracture involved the sphenoidal sinus or the middle ear region, as the table indicated. A purulent exudate was found in the middle ear cavities in four cases, blood was found in three cases but not any mention was made of the contents in nine cases. Two of the sphenoidal bony cavities contained a purulent exudate. Its presence apparently was not necessary for the development of the meningitis.

Five of the lateral composite fractures were caused by the crushing effect of an automobile wheel and anatomically were similar in all respects to the same fractures that have been described as having caused death by concussion. In these cases, apparently, the injury did not produce enough shock to cause an immediately fatal result, but the breaking of the bony sinuses eventually allowed infection to reach the meninges.

In one case of a lateral linear fracture, the ear drum was ruptured, and infection entered the middle ear cavity from the outside. In the course of time, the roof of the middle ear was eroded by the septic process, and a typical brain abscess developed in the substance of the adjacent temporal lobe by direct extension. The suppurative leptomeningitis that finally caused death was secondary to the brain abscess.

A comparatively large proportion of fractures occurred in the anterior part of the skull—seventeen. In fifteen cases, the meningitis developed from fractures which involved the frontal cavities, the eth-

moidal cells or other adjacent bony sinus. A purulent exudate was present in the frontal sinus in one instance, and several times a little extravasated blood was found. The frontal bony sinus was fractured in four cases, not only across its roof, but also in the anterior portion, and a laceration of the forehead made the fracture compound. However, the meningitis developed just as readily in the absence of purulent foci or

TABLE 15.—*Cases of Meningitis and General Sepsis*

Age Incidence

Years	Meningitis	General Sepsis	Years	Meningitis	General Sepsis
1 to 10	11	..	40 to 50	7	1
10 to 20	2	..	50 to 60	2	2
20 to 30	9	3	60 to 70	0	..
30 to 40	9	1	70 to 80	1	..
Total				41	7

Casualty Incidence

Automobile accidents	13	2	Struck by falling brick	1	..
Street car accidents	1	..	Horse kick	1	..
High falls	5	..	Knocked down in assault	1	..
Low falls	6	2	Blunt instruments	2	3
Falls down stairs	3	..	Cause unknown	8	..
Total				41	7

Duration of Clinical Course

2 days	2	..	8 to 11 days	8	2
3 days	6	..	12 to 20 days	2	1
4 days	3	..	20 to 30 days	4	1
5 days	4	..	Over 30 days	1	..
6 days	5	3	Unknown	4	..
7 days	2	..			
Total				41	7

Source of Infection in the Meningitis Cases

Source of Infection	Types of Fractures: Posterior Fissure	Posterior Composite	Lateral Fissure	Lateral Composite	Lateral Composite Crush	Anterior Fissure	Anterior Composite		
Middle ear and mastoid	6	1	5	3	3	..	..	18	Pus present in sinus 10
Sphenoidal sinus	..	..	..	1	2	..	..	3	Pus present in sinus 2
Middle ear and sphenoid combined	..	..	..	2	..	..	..	2	
External ear	..	..	1	..	..	..	..	1	Abscess temporal area, 1
Sinuses in frontal area	..	..	..	..	..	6	9	15	Pus present in frontal sinus, 1
Compound fracture	..	..	..	..	..	..	2	2	Abscess frontal area, 1
	6	1	6	6	5	6	11	41	

compound fractures, probably because the cavities in the frontal region had free communication with the outside through the nose.

In two cases the probable entrance of the infection was through a compound composite fracture of the frontal bones above the eyebrow region, in which an extensive laceration freely exposed the shattered skull. One of these persons lived for six weeks and developed a brain abscess in the frontal lobe just over a small direct laceration of the brain adjacent to the fracture. The abscess eventually eroded into the

lateral ventricle, causing a general spread of the purulent material through the cavities of the brain.

The manner in which the infection spread from the different foci to the subarachnoid space was not always obvious, and in most instances the anatomic features of the meningitis did not give any information on this point. All varieties of the lesion were described; generally purulent exudate was found involving the region of the base around the circle of Willis, or else spread in various ways over the vertex of the cerebral hemisphere. In three cases the purulent exudate was heavy and thick in the cistern around the foramen of Magendie, and a severe purulent hydrocephalus was the result. The ventricles of the brain were filled with turbid cerebrospinal fluid, and the distention caused a severe degree of intracranial tension.

In a few instances, especially around slight cerebral lacerations, a purulent membrane of varying extent was found adherent to the inside of the dura—a true suppurative pachymeningitis. The infecting organism had in some way reached the dura as well as the arachnoid, probably aided by the presence of small hemorrhage on the inside and outside of the dura, which formed an excellent culture medium for the bacteria.

Bacteriologic examinations of the meningeal exudate were made only in a few instances, but in every successful culture the bacterium was invariably the pneumococcus.

In all the cases which have been considered, the meningitis was the result of fracture of the skull. Sometimes, however, the question arose whether the presence of the infection was caused by the injury or was merely a coincidence.[3] This point was to be determined only by the clinical history. The fracture could be responsible for the meningitis only if it preceded the onset of the infection by two days or more, and was of the type that permitted the bacteria to invade the skull.

The general impression given was that the condition was insidious. The trauma was at times severe, and the diagnosis of fracture of the skull was never in doubt. Often, however, a slight injury would crack the thin bony plates which roofed the cavities at the base, and this was sufficient to allow bacteria to gain entrance. The primary effects of the violence were sometimes negligible, and it was difficult to be sure that a skull fracture was present. Accordingly, in many instances the meningitis was apparent after a lapse of several days, almost without warning, and brought about an inevitably fatal outcome.

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3. Since this paper was written, a man, aged 60, came to necropsy with a history that he had fallen down a sewer and had died a few hours after the fall. Postmortem examination disclosed a small fracture in the frontal region and a marked suppurative meningitis of several days' duration. It was probable that the fall was due to giddiness resulting from the meningitis, and that the fracture had nothing to do with the infection of the meninges, which was a spontaneous infection.

Other Septic Infections: The cases of septic infections were seven in number and were also characterized by delayed onset of symptoms, but when the condition was established, death was inevitable. The clinical course varied from six to twenty-four days.

In the four following instances the infection entered through a compound fracture of the vault, three of which were caused by assaults with blunt instruments, while one developed from a fall against an iron rail.

A white man, aged 39 years, received a compound depressed fracture of the parietal region of the vault when he was struck by the corner of a revolver butt. After a nine days' course, the case came to necropsy, disclosing a large decompression window in which the fracture had been present, an infected hernia cerebri protruding through this opening and signs of general septicemia, including a suppurative meningitis and an acute vegetative endocarditis of the mitral and tricuspid valves. A hemolytic streptococcus was isolated from the meningeal pus and from the splenic pulp.

Another patient, a colored man, aged 29 years, who had received a small compound depressed fracture in the left frontal region, lived six days. He died during the course of a decompression operation performed shortly after his admission to a hospital. Necropsy disclosed the fracture and the way in which the bacteria gained entrance through the hole in the scalp and skull. Just beneath the broken bone the dura was intact, but there was a small extradural collection of purulent fibrin in this area. In addition, there were signs of general septicemia, which comprised an extensive purulent pachymeningitis and leptomeningitis and hemorrhagic nephritis. A bacteriologic examination was not made in this case.

One case of *Staphylococcus aureus* septicemia was described, following a compound depressed fracture in the left parietal region. A craniotomy was performed, and the patient survived only thirteen days. Necropsy showed an infected scalp wound and infected dura near the decompression site, with extension of septic thrombi through the smaller vessels of the dura to the right lateral sinus. The lungs were filled with huge embolic abscesses and septic infarcts. The picture was typical of staphylococcus infections, and the organism was cultivated from the abscesses of the spleen and of the lung.

In the fourth case there was a compound comminuted fracture in the right parietal region on which an operation was not performed. The patient lived for nine days. At necropsy, there was a collection of purulent fibrin which accompanied the middle meningeal vessels on the right side in a dentritic pattern to the foramen spinosum. There was no infection of the inside of the dura or brain, but there were signs of general septicemia, such as abscesses of the kidney and abscesses in the left psoas muscle and left elbow. The body was embalmed, and it was not possible to make a bacteriologic examination.

The other cases were the result of a fall in one instance and of automobile injuries in the other two. The fractures were not compound, but involved the middle ear region, and it was from this bony sinus that the septic infection originated.

One man fell from a ladder and sustained a posterior linear fracture which extended into the right mastoid cavities. After twenty-four days he died as the result of a septic thrombosis of the right lateral sinus, with multiple septic infarcts in the lungs.

The two automobile accidents caused lateral fractures which shattered the middle ear region. One man developed a huge foul abscess between the dura and the skull of the middle fossa, with marked necrosis of the mastoid area, but abscesses were not found elsewhere. In the other case in which there was a similar but less extensive fracture, a marked suppurative pachymeningitis interna was seen near the region of the fracture, but leptomeningitis or any other anatomic signs of infection were not present. Both these persons lived about six days.

The essential factor in all these cases was that the fracture permitted the entrance of the microorganism through the skull, and that conditions were such that its spread was further facilitated. Any small intracranial extravasations of blood, especially small epidural clots under a compound fracture, made a splendid nidus in which pathogenic bacteria could grow, and when they reached such a lesion, the invasion was established definitely.

TABLE 16.—*Deaths Caused by Operative Procedures*

Age, Color, Sex	Casualty	Fracture	Procedure	Cause of Death
White woman 46 years	Low fall	Anterior linear fracture	Given ether anesthetic for a Colles fracture of left wrist	Ether anesthetic; died during anesthetic
White boy 14 years	Automobile	Posterior linear fracture; direct and contre-coup brain laceration	Bilateral decompression for intracranial pressure; operated on day of injury	Postoperative shock; died a few hours after operation
White man 68 years	Automobile	Composite vault fracture; direct and contre-coup laceration; fracture of ribs and left clavicle	Decompression in area of comminuted fracture; operated on day of injury	Postoperative shock; died a few hours after operation
White man 21 years	Automobile	Compound lateral fracture with tear of superior longitudinal venous sinus	Decompression to stop hemorrhage from venous sinus; operated on day of injury	Sudden death from hemorrhage during operation

Deaths Caused by Operative Procedures: Postoperative Shock.—This group included four cases; three of the patients died because of the operation on the skull, while one died under ether anesthesia. The data are given in table 16.

The first three cases do not require detailed comment.

The fourth, however, was the only example in the series of an epidural hemorrhage which resulted from laceration of the superior longitudinal sinus. The fracture as first noted during life was a compound depressed fracture on the right of the midline over the coronal suture. During the removal of the bony fragments at operation, it was discovered that the superior longitudinal venous sinus was torn by the sharp edges of the fracture, and that one of the tributary veins on the inside of the dura was similarly torn. These bleeding points were rapidly sutured, but the patient lost so much blood during the procedure

that he died on the table. At necropsy, the decompression window was found in the region already noted, with a linear fracture on its left extending down the coronal suture to the left middle fossa, while another linear fracture on its right ran diagonally in a posterior direction to the right posterior fossa. The fracture had been produced by severe violence, for on the vault the posterior edges of the fracture were displaced to the left of the anterior edges. The sinus and its tributaries had been sutured, but the general pallor of the body indicated that much blood had been lost. The brain showed a laceration at the site of the decompression, about 1½ by 2 inches, and there was a hernia cerebri present in the opening through the skull. Contre-coup lacerations of the brain were not found.

In the series of 507 cases, thirty additional trephine operations were performed, but, while they did not help the patient materially, there was not any evidence to indicate that they were responsible for the fatal termination. Thirteen were tried in cases included in the subdural hemorrhage and lacerated brain categories, seven in epidural hemorrhages, four in cases of meningitis, four in cases of general septic infection and two on patients who died of pneumonia. Relatively little information, however, was furnished by these unsuccessful operations, and the advisability of the procedure was best considered among the cases studied clinically.

Deaths from Epileptic Convulsions.—Death from epileptic convulsions was diagnosed in three cases. The patients had received a fracture of the skull a year and a half to two years prior to death and had recovered from the immediate effects of the injury. Two of the fractures were compound composite fractures in the frontal region of the vault, which had caused an extensive direct laceration of the adjacent frontal lobe of the brain. An operation had been performed, and at necropsy the brain was found firmly adherent to the decompression window by a pigmented scar. The third case showed an old healed depressed fracture in the left posterior parietal region, while the lateral frontal region on the right was extensively adherent to the dura over a wide area by pigmented scar tissues, which was obviously the result of an old contre-coup laceration in this region.

The data are given in table 17.

In each case there was a latent period of several months after the immediate effects of the injury were entirely over, and the patient was discharged as cured. Then convulsions began and grew worse until death ensued. The adhesions obviously had some association with the convulsions, but the nature of their influence was not evident. The etiology of this type of epilepsy was just as obscure as that of idiopathic epilepsy.

Deaths Caused by Injuries Elsewhere in the Body.—Thirty patients showed fractures of the skull of moderate degree which were accompanied by severe injuries in other portions of the body. Death was obviously the result of the latter.

TABLE 17.—*Cases of Epilepsy*

Age, Color, Sex	Casualty	Lesions of Skull at Necropsy	Lesions of Brain	Cause of Death	Duration after Primary Injury
White man 45 years	Low fall	Decompression window, right frontal region	Huge adhesion of right frotal lobe to site of operation	Generalized epileptiform convulsions	2½ years
Colored man 52 years	Assault with hatchet	Decompression window, left frontal region	Huge adhesion of left frontal lobe to old operative area	Generalized epileptiform convulsions	1½ years
White man 68 years	Unknown	Healed depressed fracture, left posterior region	Contre-coup laceration of right frontal lobe adherent to adjacent dura	Epileptiform convulsions	Unknown, but of some duration

TABLE 18.—*Cases of Injuries Elsewhere in the Body*

Casualty Incidence			
	No. of Cases		No. of Cases
Automobile accidents	24	Low fall	3
Street car accidents	1	Unknown	1
High fall	1		
Age Incidence			
Years		Years	
1 to 10	4	40 to 50	3
10 to 20	4	50 to 60	3
20 to 30	1	60 to 70	2
30 to 40	9	70 to 80	4
Duration of Clinical Course			
Found dead	2	12 to 24 hours	1
Minutes	10	2 to 3 days	1
1 to 6 hours	10	6 days	1
6 to 12 hours	4	25 days	1
Location of Fractures			
Posterior linear	11	Anterior linear	5
Lateral linear	10	Vault linear	1
Lateral composite	3		
None of these cases showed any intracranial lesions of any severity			
Causes of Death			
Shock from severe fractures of spine, ribs, pelvis or bones of extremities			12
Internal hemorrhage in chest or abdominal cavity from ruptures of liver, spleen or lungs			16
Septic infections from injuries of trunk			2

Deaths Due to Natural Causes Unrelated to Fractures of the Skull.—There were twenty-five such cases, and all showed slight fractures of the skull. Death was obviously the result of some other condition which did not have any connection with the fracture of the skull.

In six of the cases there were old healed fractures of the skull, easily identified as such at necropsy. Only a small proportion of the healed fractures were found occasionally in the routine postmortem examinations. All of them showed old healed lacerations of the brain of the contre-coup or direct type. None had been subject to operation, and the primary injury had cleared up more or less spontaneously. It was probable that a large proportion of patients with less severe fractures of the skull did recover in this fashion, although it was difficult to determine the exact percentage with any degree of accuracy.

Most of the patients had had a severe general illness at the time the injury was received, and the fracture was merely a minor factor

TABLE 19.—*Deaths Due to Natural Causes*

	No. of Cases		No. of Cases
Casualty Incidence			
Automobile accidents	5	Assaults by blunt instruments	2
Low falls	6	Unknown	11
Fall down stairs	1		
Age Incidence			
10 to 20 years	1	50 to 60 years	5
20 to 30 years	3	60 to 70 years	3
30 to 40 years	8	90 to 100 years	1
40 to 50 years	4		
Duration of Clinical Course			
Found dead	4	Days	4
Minutes	7	Old fractures	6
Hours	4		
Location of Fractures			
Posterior fissure	4	Anterior linear	7
Lateral fissure	6	Anterior composite	3
Lateral composite	3	Vault linear	2
Causes of Death			
Spontaneous cerebral hemorrhage	3	Illuminating gas poisoning	1
Acute alcoholism	7	Pulmonary tuberculosis	2
Chronic alcoholism	1	Erysipelas	1
Chronic meningitis	1	Coronary artery disease	3
Chronic encephalitis	1	Rupture of aortic aneurism	1
Idiopathic epilepsy	1	Lobar pneumonia	2
		Obstruction of bile ducts	1

in producing the fatal result. It is probable that some of the brain conditions caused symptoms of vertigo and unsteadiness, so that the deceased was made to fall and fracture his skull in this fashion. This was probably true of the three patients who died of spontaneous cerebral hemorrhage.

Fractures of the Skull in the New-Born and in Infants Under 5 Months of Age.—The skull of the young infant is entirely different in structure from the skulls of older children and adults. While the cranial bones of the latter are fused, forming a compact rigid case for the brain, the skull of the infant is a pliable sac of bony plates held together by a membrane, so that it is easily distorted by violence. Fractures of the skull in the early months after birth, therefore present peculiarities not characteristic of the lesion in the later periods of life.

In the infant, the base of the skull is small in proportion and closely knit and elastic. The vault is relatively voluminous and is composed of five curved, easily bent plates of bone, comprising the two parietal bones, the two halves of the frontal bone and the occipital bone. The dura lines these segments rather loosely, except at the edge of the segment, where it is firmly attached. The bony plates are joined by a thick fibrous membrane which is widened in the midline at each end of the two parietal bones to form fontanels. The larger anterior fontanel is placed at the coronal suture and the smaller posterior at the lambdoid suture.

The parietal bones are most often fractured because they are the most prominent segments of the vault and, therefore, the area most subjected to trauma. They were involved in all five cases of this series, and in all

TABLE 20.—*Fractures of the Skull in Young Infants*

Age, Sex	Casualty	Duration	Fracture	Other Lesions	Cause of Death
New-born; girl	Unknown	2 days	Linear parietal fracture	None	Concussion
3½ months; boy	Hit by thrown pitcher	2 days	Comminuted parietal fracture	Direct laceration of brain	Effects of concussion
17 hours new-born; girl	Thrown from window	17 hours	Linear parietal fracture	Direct laceration of brain	Effects of concussion
3½ months; boy	Unknown fall?	Unknown	Linear parietal fracture	Direct laceration; contre-coup lacerations frontal lobes; bilateral subdural hemorrhage, 10 Gm.	Cerebral compression
4 months, 4 days; boy	Thrown to floor of taxi	3 hours	Comminuted parietal fracture	Direct laceration; tear of membrane of anterior fontanelle	Concussion

except one, the fractures were confined to this region. In the other case, an extensively comminuted fracture, the frontal bone and part of the anterior fossae were included, together with the parietal plates. This, however, was rather unusual, for generally the base of the skull escapes injury in infants. The membranous attachments of the vault and the mobility of its plates have a tendency to confine the effect of the violence to the bones primarily struck, with but little transmission of the force to other parts of the cranium.

The fractures in the parietal bone depend on the directions of the bony striations which radiate out from the central prominence or boss toward the edge of the bone. The linear fractures occur in the direction of these striations. They appeared as simple straight splits of the bone from the edge to the central prominence in two cases, and in a third the fracture was angular, with the point of the angle at the apex of the prominence. The other two fractures were comminuted extensively.

Lacerations of the brain occurred in four cases as direct lacerations in the region of the fracture, while contre-coup lacerations on the under surface of the frontal lobes occurred only once. In one instance, lacerations of the brain were absent. As a rule, the cerebral lesion was not productive of complications, except in one infant who developed a subdural hemorrhage.

The dura was sometimes torn by the bone, but not invariably, and lacerations of the brain were readily produced without a dural tear.

A tabulation of these cases is given in table 20.

Apparently the cerebral concussion played the most important rôle in causing death. In infants, as in elderly people, however, a slight trauma often had the effect of depressing the central nervous system only to a slight degree, but the organism did not possess enough vitality to throw off the effects of the primary shock altogether. They usually remained for several hours or days until death ensued.

The subdural hemorrhage caused death by compression and, from the postmortem appearance of the blood, had evidently been of many days' duration; the lack of reliable history, however, was a handicap to study of the case.

SUMMARY

1. Five hundred and twelve cases which came to necropsy were reviewed in order to study the different conditions that caused death in fractures of the skull.

2. The types of fractures were classified as

A. Fractures of the vault and base
- (1) Posterior fractures
  - (*a*) Fissure fractures
  - (*b*) Composite fractures
- (2) Lateral fractures
  - (*a*) Fissure fractures
  - (*b*) Composite fractures
- (3) Anterior fractures
  - (*a*) Fissure fractures
  - (*b*) Composite fractures

B. Fractures of the vault
- (1) Fissure fractures
- (2) Composite fractures
- (3) Depressed fractures

3. Five hundred and seven cases occurred in children and adults whose ages ranged from the first decade of life to the tenth decade. In this group, the complications which caused death were considered in detail.

4. Death in 139 cases was caused by cerebral concussion. The majority of these patients died in the first hour following the trauma, and all succumbed inside of the tenth hour. The fractures were, for the most part, severe and extensive.

5. Death from exhaustion occurred in fourteen instances. The injured persons did not die immediately, but they were unable to rally against the primary shock, finally dying after periods varying from one to twenty days. In one case, extensive contusions in the motor area of the right cerebral cortex induced violent convulsions which finally brought about death from exhaustion.

6. Death in twenty-seven cases was caused by a terminal lobular pneumonia, which as a rule set in from two to nine days after the injury.

7. A large number of patients succumbed to cerebral compression, caused by subdural hemorrhage in 132 instances, and to extensive lacerations of the brain in twenty-four instances.

(*a*) The majority of subdural hemorrhages were the result of contre-coup or direct lacerations of the cerebral cortex. A few were produced by the tearing of tributary veins from the brain to the superior longitudinal sinus, and one was caused by the tearing of an old adhesion between the brain and the dura. In one case, laceration of the dura and a branch of the middle meningeal artery caused the artery to bleed profusely into the right subdural space.

(*b*) The fatal lacerations of the brain were either surface lesions which covered a wide area of the cerebral cortex or big cavities in the brain substance which contained a large quantity of blood. Some of these caused extensive hemorrhages in the ventricles of the brain. None of the lesions, however, were accompanied by a subdural hemorrhage of any size.

There was reason to believe that many of the cases in this group showed a moderate grade of edema of the brain.

8. Sixty-one patients died of cerebral compression caused by an epidural hemorrhage. Most of the hemorrhages were the result of a laceration of the middle meningeal artery by the fractured bone. The posterior branches of the middle meningeal were involved much more frequently than the anterior branches. A few hemorrhages were caused by lacerations of the lateral sinus. One hemorrhage arose from a laceration of one of the anterior meningeal arteries.

9. The interval between the casualty and the death varied considerably in the cases of cerebral compression. Most persons succumbed during the first twenty-four hours, but others lingered for from eight to fourteen days.

10. Acute suppurative leptomeningitis was responsible for the death of the patients in forty-one cases. Fractures across the bony sinuses

of the skull caused the infection in the majority of instances, although in two cases the organism entered through a compound fracture.

11. Seven persons died of various other septic infections caused by the fracture of the skull. The infective agent gained entrance through compound fractures and through fractures involving a bony sinus. Two of the patients developed a typical *Staphylococcus aureus* septicemia with thrombosis of the lateral sinus and septic infarcts in the lungs. One person died of a general septicemia caused by the hemolytic streptococcus. Other patients developed large collections of pus in the epidural and subdural spaces.

12. Patients who died of suppurative meningitis or other form of sepsis lingered for from several days to several weeks. The infections were insidious in their onset and, once established, were inevitably fatal.

13. Four patients died as the result of an operation on the skull. One woman died under ether anesthesia. Two patients died as the result of postoperative shock. One man died during the progress of the craniotomy, death being caused by a profuse hemorrhage from the superior longitudinal sinus, which had been lacerated by the fracture.

14. Three persons died as the result of a traumatic epilepsy, which was associated with an old extensive adhesion of the brain to the dura, mainly in the frontal region. The convulsions usually set in several months after the primary symptoms of the fracture had subsided, and the patients survived from one and one-half to two years after the injury.

15. Thirty patients died as the result of injuries in other parts of the body which were caused by the same violence that fractured the skull.

16. Twenty-five patients died as the result of a condition which did not have any connection with the trauma.

17. Five of the 512 patients who came to necropsy were new-born infants or infants under 5 months. The skull at this age is not a homogeneous bony case, but it is composed of bony plates held together by tough membrane. This peculiarity served to confine the fracture to the bones primarily struck, which in most cases were the parietal. The cause of death was usually concussion or exhaustion, although one baby developed a subdural hemorrhage from a direct laceration of the brain.

PART II. CLINICAL ASPECT OF COMPLICATIONS PRODUCED BY FRACTURES OF THE SKULL[4]

The clinical records are from the wards of the first surgical division of Bellevue Hospital, and cover a period of five years, from 1919 to 1924. Sixty-one cases are presented in detail. These comprise cases of

4. Many of the fatal cases in this series were included in the necropsy material in part I, but, as the two series were considered separately, there was no duplication.

fatal fractures of the skull in which a craniotomy had been performed, and cases of fractures from which the patient recovered both after operation and under expectant treatment. All that survived were placed under observation after leaving the hospital, either until the patient was considered to have recovered from the effects of the injury or until he could no longer be found by the "follow-up".

A number of cases in which craniotomy was not performed and the patients died are not included, as the clinical histories in all such cases were similar and can be discussed adequately in a few words.

For the sake of convenience, an arbitrary division of the clinical material is made as follows: (A) fatal cases in which craniotomy was not performed. (B) operative cases, fatal and nonfatal. (C) fractures of the skull from which the patients recovered under expectant treatment.

FATAL CASES IN WHICH CRANIOTOMY WAS NOT PERFORMED

Fractures of the skull from patients who died without having an operation performed were of two varieties.

The patients who had the first type were admitted in a moribund condition, suffering from profound cerebral concussion caused by a recent trauma; or they had been injured several hours or days before they reached the hospital and were in the last stages of cerebral compression or meningitis. The clinical signs often did not give any indication of the nature of the intracranial condition. The patients were in a state of profound prostration, shown mostly by deep coma, complete insensibility, flaccidity of all muscles, stertorous respiration, pupils fixed and not responsive to light and a pulse and temperature variable in reaction. The poor physical condition of the patient was a clear contraindication against a craniotomy; in addition, there was not anything to suggest that the operation would have been of any avail.

The patients who had the second type were admitted to the hospital showing few signs of injury and often perfectly conscious and rational. They usually lived for several days, and during their stay in the ward did not develop any signs or symptoms which encouraged the surgeon to operate. They finally lapsed into coma and died. At necropsy, death was found to be due to causes of such insidious onset as an acute suppurative meningitis, general septic infection or terminal bronchopneumonia.

OPERATIVE CASES

In deciding on the advisability of operation, the clinician must answer two questions. 1. Do the clinical observations indicate a fracture of the skull? 2. Are the signs definite enough to identify an intracranial condition which will be benefited by craniotomy?

Diagnosis of the Fracture.—The presence of a fracture of the skull was determined in several ways—by the history of the trauma, by the general condition of the patient, by the external signs of the injury of the head, by examination of the spinal fluid and finally by roentgen-ray examination of the skull.

(*a*) History of the Trauma: Information concerning any injury which the patient might have received always helped in making a diagnosis. In the majority of cases this information was available, although a few persons were admitted to the hospital in an unconscious state without any previous history. The chief value of this information lay in the fact that it gave the clinician a suggestion of what investigations he should pursue.

(*b*) General Condition of the Patient: If there were definite signs of well marked cerebral concussion or cerebral compression, the suggestion that the patient was suffering from some intracranial injury and perhaps from a fracture of the skull could be entertained with reasonable certainty.

As has been noted, concussion occurred immediately after the trauma, and the patient appeared in a state of prostration and coma which lasted for a variable period of time. Sometimes it was of long duration, and the patient was unconscious for days; in other cases, it passed away within the hour, and the patient recovered his faculties completely. In rare instances, the fracture occurred without any signs of concussion. Generally, on admission to the hospital, the patient presented cerebral symptoms of moderate severity and was described as stuporous or easily aroused, irrational, mentally confused and irritable, often complaining of vertigo, headache and vomiting.

Cerebral compression was often present on admission, but it was not always easy to recognize it as such until many clinical tests had been tried, especially when deep coma had already begun. The most typical instances occurred in patients who were admitted to the hospital in a rational condition and then lapsed gradually into unconsciousness, complaining of increasingly severe headache, vertigo and vomiting, with slowing of the pulse rate and a rise of blood pressure. Unfortunately, the patient was not always observed at this characteristic stage, and then the symptomatology gave little assistance. Reliance had to be placed on a study of various signs, which included the pulse and blood pressure records, the pressure of the spinal fluid, examination of the eyegrounds and a consideration of the various neurologic signs.

The pulse and blood pressure readings, while important, had to be interpreted with some degree of caution. So many factors influenced the circulatory system that neither the pulse nor the blood pressure reacted to intracranial conditions with any degree of consistency.

In a typical case of cerebral compression the pulse became slow, dropping from a rate of from 70 to 90 a minute to about 40 to 60 a minute. It was then described as a slow and bounding, or as a "high tension," pulse. Occasionally it was rapid and weak, and its rate was as high as 120 a minute. Its value as a diagnostic sign was, on the whole, rather limited.

The blood pressure readings also varied. A high blood pressure of systolic 180 and diastolic 80 or one of systolic 230 and diastolic 90 indicated definitely a severe grade of intracranial pressure.

However, many patients showed comparatively low pressures, such as systolic 128 and diastolic 80, and the like. It was not infrequent to find a great variation in the blood pressure records in the same patient when they were taken at different times. The observations were not consistent enough nor were the readings taken frequently enough to allow definite conclusions to be formulated; but apparently during the first few hours of the clinical course, the systolic blood pressure was high, and in the later stages it tended to drop as much as 40 or 60 points. This suggested that the circulation either was adapting itself to an abnormal condition or was weakening under the strain. It also indicated that the readings had to be taken at frequent intervals in order to convey information of significance.

In some instances, the spinal fluid was blood-stained and flowed out under pressure. This was a reliable sign of intracranial tension, but the phenomenon was recorded only as a qualitative observation. A manometer was not available at the time the patients in this series were in the wards, so quantitative measurements of spinal fluid pressure were not made. There was, therefore, no criterion by which to compare the degree of pressure in the different patients.

Ophthalmoscopic examination of the eyegrounds revealed circulatory disturbances in that region and so gave valuable information concerning intracranial pressure. In cases of moderate severity, the vessels of the disk were engorged, the disk appeared hazy or in some cases definite papilledema was found. In several instances, the opthalmoscopic examination showed that the eyegrounds were affected more on one side than on the other, and generally the cause of the increased pressure was located on the side of the more involved eyeground.

The neurologic examination was always necessary in any case of injury of the head, especially if a craniotomy was under consideration, for it often located the point of disturbance with a fair degree of accuracy. These tests included a routine determination of the following points: (1) conditions of the pupils and the oculomotor system; (2) conditions of the facial and tongue muscles; (3) superficial reflexes of the trunk; (4) deep reflexes of the extremities; (5) abnormal neurologic reactions, such as patellar and ankle clonus, Babinski and Kernig sign,

stiff neck and others; (6) abnormal conditions of muscular groups, such as weaknesses, spastic and flaccid paralysis and convulsive seizures.

1. The reaction of the pupil to light was determined, it being especially noted whether the reflex was absent or sluggish. If the patient was at all rational, the test for accommodation was tried. The information so obtained was generally inconclusive, except that a sluggish or immobile pupil indicated that the intracranial complications were probably severe.

In several instances the pupils were unequal, one was dilated, while the other was contracted, and this sign seemed to point to a lesion definitely located on one side of the cranial cavity. In seven fatal cases in which there was a subsequent necropsy, the right pupil was observed to be much larger than the left. Five of these showed an epidural hemorrhage on the right, one a subdural hemorrhage on the right and one an epidural hemorrhage on the left. The cause of the inequality was not easy to determine, but the difference in the size of the pupils depended, apparently, on the unilateral nature of the intracranial condition.

Abnormalities of the oculomotor muscles, such as deviation of the eyes to the right and the head to the left and lateral movements of the eyeballs or nystagmus, were also noted. This, in all probability, was caused by cortical irritation, but the explanation was not obvious.

2. Paralysis and weakness of the facial muscles on one side and deviation of the tongue to one side were noted, but, beyond indicating some sort of intracranial disturbance, their significance was uncertain. Many of these phenomena were transitory and cleared up finally.

3. The superficial reflexes of the trunk, namely, the abdominal and cremasteric reflexes, were occasionally active and equal, sometimes absent on both sides, and at other times more active on one side than on the other. These reflexes apparently became less and less active as the intracranial condition increased in severity, and thus varied with the condition of the patient, but their value as a guide to cerebral involvement was limited.

4. The various deep reflexes, including the biceps, triceps, patellar and Achilles' tendon reflexes, also varied, from being equal and hyperactive or equal and sluggish to being markedly unequal, when one side was much more active than the other. This generally indicated a unilateral intracranial lesion, but did not always indicate the side of the brain involved nor the nature of the lesion causing the trouble. The presumption was that the cerebral cortex was affected in some way.

5. The abnormal reflexes, such as patellar clonus, Babinski's reaction and several others, also indicated cortical involvement of some kind, generally of considerable severity. Much significance was attached to these abnormal reflexes when they were more pronounced on one side than on the other, and the inference was that the side which reacted

the strongest referred its behavior to the corresponding motor area of the cerebral cortex, which had been injured.

The occurrence of an abnormally rigid neck and a positive Kernig sign was generally referable to a suppurative leptomeningitis, but was also caused by subarachnoid hemorrhages, which could also produce cortical irritation. The only way in which differentiation could be made with any degree of certainty was by examination of the spinal fluid.

6. The presence of weakness, paralysis or convulsions in various muscle groups, however, was a sign of great importance, and will be discussed at greater length later. The lesions giving rise to these abnormalities were various and could be located fairly definitely over some portion of the brain. As a rule they indicated involvement of certain areas of the motor cortex, always on the side of the brain opposite to the paralyzed muscles. In some instances the lesion was a direct laceration of the motor area, in others it was a depressed fracture impinging on the cortex and in many it was an epidural or subdural hemorrhage pressing in the brain substance. The definite nature of the clinical sign usually suggested the site where a craniotomy would be most likely to relieve the intracranial complication.

(*c*) Signs of External Injury: The external signs of injury of the head were always important, because through them the fracture could be located with a reasonable degree of certainty in a large number of cases. At least, they indicated where violence had been applied to the head and so suggested what intracranial conditions might logically follow the trauma. Injuries of the scalp were important in this respect.

The scalp was particularly vulnerable to the violence, as it consisted of a layer of skin applied closely to the underlying hard cranium. Impact against a projection caused lacerations of varying length and depth, which were linear or stellate if the force was applied at right angles to the skin, but which resulted in a flap of skin if the projection impinged on the scalp tangentially. Occasionally a fracture of the skull was seen at the bottom of the laceration.

If the head came in contact with a smooth surface, the scalp injury was not always obvious. Direct impact, however, at times produced a contusion of the superficial layers without laceration. Sometimes a lesion of this kind appeared as a mound with a softened area in the center, where the force had turned the fat into pulp. A tangential impact produced abrasions of different degrees, sometimes associated with a contusion. In a few cases, the violence did not produce skin lesions of any kind; generally the force was a relatively slight one, and the skull was thin.

In some instances, a local lesion was not evident on the surface of the skin but a huge, doughy hematoma of the vault was often present, causing the scalp to bulge perceptibly in certain regions. Investigation

showed that much extravasated blood was present in the galea or was separating the galea from the skull. The hemorrhage was often caused by an extensive fracture which admitted blood through the broken bone from the inside of the cranium. In cases in which the dura and brain were lacerated, traumatized brain tissue was often found with the blood. In two cases, hematomas of this kind were observed to increase in size while the patient was in the ward, and thus served to establish the site of the fracture with a reasonable degree of certainty.

In some instances in the necropsy material, fractures resulting from the crushing action of an automobile wheel were sometimes accompanied by separation of the scalp from the bone, without an external tear of the skin. The grinding action of the wheel caused the separation and produced a hematoma from the torn vessels near the junction of the scalp and the skull. This lesion was found mainly on the lateral part of the head. In several instances, however, the scalp was torn rather extensively by the violence, especially in the region of the ear.

Among the sixty-one cases, injuries of the scalp occurred as follows: lacerations with compound fractures, 12; lacerations, 18; contusions and hematomas, 16; abrasions, 3; no scalp injuries, 12.

Five of the twelve cases of uninjured scalps did not show demonstrable external trauma of any kind. This point was observed many times in the necropsy material and indicated that a serious fracture of the skull was not necessarily accompanied by surface injuries.

Other signs of external injury were hematomas of the eyelids, bleeding from the nose, mouth and ears and vomiting of blood from the stomach.

The hematomas of the eyelids were of different degrees, varying from a contusion of small size to huge swellings in both lids, sufficient to close them. They involved either one side or both and also were confined to either the lower or the upper lid, or they involved both. In many cases the ecchymosis was caused by rupture of one of the superficial blood vessels in the loose lid tissue and was not associated with fracture of the skull. In other instances, there was a fracture of the orbital plate, and the hemorrhage arose from rupture of a vessel inside the orbit, which bled through the fracture. The orbital lesion was generally the result of violence applied to the frontal region, but occasionally occurred as the result of a severe trauma applied to another portion of the skull.

In the series of sixty-one cases, hematomas of the eyelids occurred five times on the right side, six times on the left and once on both sides. As a rule, this sign merely indicated some kind of an injury and did little more than suggest the presence of a fracture.

In one instance there was a condition of subcutaneous emphysema in the frontal region, associated with ecchymosis in the eyelids. This was

attributed to a fracture of the frontal sinus with a tear of the mucous membrane and the entrance of air into the subcutaneous tissues by efforts of respiration.

Bleeding from the nose and mouth was mentioned four times, but the sign did not have any great significance. It occurred often without a skull fracture, resulting from some injury to the mucous membrane. Occasionally, in cases of severe hemorrhage, it indicated that a fracture involved the bony cavities communicating with the nose, such as the ethmoidal, frontal or sphenoidal sinuses. A posterior fracture rarely produced such a hemorrhage by lacerating the mucous membrane of the pharynx.

Inhalation and swallowing of blood also resulted from these hemorrhages. With the patient in coma and blood flowing into the upper respiratory passages, it was not unusual for a small quantity to be aspirated. At necropsy, these patients showed irregular, dark red, sanguineous areas in the pale lung tissues, easily identified as inhaled blood by the lobular distribution. It is probable that this condition might later have encouraged the development of a lobular pneumonia.

The blood was sometimes swallowed and at necropsy was encountered in the stomach in the form of fresh blood clots or as transformed blood with a rusty red or a brown coffee color. Clinically, the patients sometimes vomited the blood. In the sixty-one cases, this occurred eight times, six cases showing fresh blood in the vomitus, while two patients exhibited transformed blood.

One sign to which considerable significance was attached was bleeding from the external ear canal. This was described eleven times in the series of sixty-one cases, three times from the right ear and eight times from the left.

In some of the cases that came to necropsy, the bleeding ear canal was investigated thoroughly to determine the source of the hemorrhage. Many showed a fracture of the lateral portion of the skull, which descended along the squamous portion of the temporal bone, entered the upper portion of the circumference of the canal and then ran into the middle fossa in various directions. Some of these fissures passed directly across the roof of the middle ear cavity and ruptured the drum in various ways, but others ran well forward of this structure and left the drum intact. The cases with intact drums usually bled from a laceration of the lining membrane of the external ear, directly induced by the fracture. When the drum was ruptured, the hemorrhage possibly arose from the trauma to the middle ear as well as from the injury to the external ear.

In one instance—a street car accident—fracture of the skull was not present on the side of the ear hemorrhage, but investigation of the structures of the ear showed lacerations and contusions of the membrane

of the external canal, while the drum was intact and the middle ear free from blood. There was a fracture on the opposite side of the skull, but this did not have any connection with the bleeding auditory meatus. This seemed to suggest that a fracture was not always the cause of the hemorrhage. However, cases of this kind were exceptional, and in the majority of instances, bleeding from the ear canal was a reasonably certain indication of a fracture of the skull in that region.

(*d*) Examination of the Spinal Fluid: The spinal fluid obtained by lumbar puncture was examined in forty-seven of the sixty-one cases. Bloody fluid in all three tubes was present in thirty-nine, and, of these, fourteen recorded as under pressure. Two times the fluid was turbid, four times it had a yellowish tinge and two times it was clear. In fourteen cases there was not any record of lumbar puncture.

The bloody fluid merely indicated that there was a hemorrhage into the subarachnoid space or into the cavities of the brain from some cause, but it did not throw any light on its etiology. Both cerebral lacerations and spontaneous cerebral hemorrhages cause sanguineous lumbar punctures, so that the sign had only a limited value in differential diagnosis. Without doubt, laceration of the brain was responsible for the blood in the majority of cases considered here. The fluid often flowed out under pressure, especially during the hours immediately following the trauma.

If the patient survived a few days, the character of the fluid changed. First the color was a bright blood red, later a turbid brownish red, still later a clear brownish or yellowish color; finally, the normal clear, watery type would appear. These transformations correspond roughly to the different changes which took place in the laceration of the brain and indicated different stages in the transformation of the extravasated blood until its final disappearance.

The spinal fluid also indicated the onset of suppurative meningitis. At first, after varying intervals of several days following the injury, there would be a tremendous increase in the quantity of the fluid. Later it became cloudy and finally purulent. Microscopic examination at this stage generally showed polymorphonuclear leukocytes and sometimes bacteria.

(*e*) Roentgen-Ray Examination: The roentgen ray was used in twenty-nine cases as an aid in the identification of the fracture. Four of these were craniotomies, and the examination was of great value in locating the bone lesion before the operation was performed. The other twenty-five cases were fractures from which the patient recovered under expectant treatment, and in all of them the diagnosis was made only by this means. More will be said about the applicability and the limitations of the roentgen-ray examination when these cases are discussed.

Indications for Craniotomy.—The next point to determine was whether or not a craniotomy was necessary. In this series, thirty-four patients were subjected to this operation, and of these twenty-three died and eleven recovered. In every case there was a definite indication for the operation, which could be classified as follows: (*a*) compound or depressed fractures—fatal cases 3, recovery 7, total 10; (*b*) increased intrancranial pressure—fatal cases 20, recovery 3, total 23; (*c*) local paralyses—fatal cases 0, recovery 1, total 1.

(*a*) Compound or Depressed Fractures: The reasons for surgical intervention in cases of compound fracture are obvious enough. Laceration of the scalp exposes the broken bones, and often hair and other infective material are present in the depths of the lesion. Conditions are right for the onset of a septic infection, and unless the fractured area is cleaned, death almost surely will result.

Depressed fractures are also sufficient in themselves to justify a craniotomy, even when the fracture is not compound. The bony fragments impinge on the adjacent portion of the brain, where they often cause direct lacerations of the cortex and sometimes produce definite disability.

The causes of the casualties in my ten cases were as follows: blunt instruments, 3; high falls, 3; falling objects, 2; automobile accidents, 1; bullet wound, 1. The trauma was such that an object with a limited area of impact in some way came into contact with the skull, causing a more or less localized lesion.

The ages of the patients showed two boys aged 14 and 15 years, four men aged 26 to 30 years, two men aged 44 years and two men aged 50 and 55 years.

The depressed portions of the fractures were situated in the left parietal region in three cases, in the right parietal in two and in the frontal region in five. A laceration was present in every instance, and in nine cases exposed the fracture. In one instance, the laceration did not make the fracture compound.

In the three fatal cases, hair and other foreign material were driven in among the fractured bones in two instances, while in the other case the bone was exposed freely. All died of an acute suppurative meningitis. Two were operated on early, and the infected material was removed, but the infection made its appearance on the third day, and death followed on the fifth. The other patient did not give any indication that he was in danger, but on the eighth day signs of meningeal irritation developed. Operation disclosed a severe frontal fracture with hair in between the bones and the septic process already present; he died on the ninth day.

The patients that recovered probably escaped because the infection was kept well under control. Six of these fractures were definitely compound. Two were operated on shortly after admission, and in one

of these hair was found between the broken bones (case 3). Trephine was performed on one patient on the second day over a compound fracture in the right parietal region, and later a craniotomy was performed in the left parietal region because the hematoma of the scalp began to increase in size at that point (case 4). One boy, aged 14 years, developed an infected compound fracture in the left frontal region, but the craniotomy on the fifth day served to stay the infection (case 5).

Another man, aged 28, fell down the hatchway of a ship and sustained a severe compound fracture of the frontal region, which extended into the frontal sinus. The operation was performed eight days later, and the patient recovered without any sequelae (case 2).

One boy, aged 14 years, had a fragment of bullet removed from the frontal region fourteen days after the shooting and also recovered without incident. This, however, was not remarkable, as the foreign body penetrated only the outer table and did not involve the inner table (case 6).

A man, aged 44, was operated on for a depressed fracture in the left parietal region which, however, was not compound. The fracture apparently produced definite weakness and convulsive jerking of the right leg; craniotomy on the fifth day relieved these symptoms in part (case 7).

Three of these cases produced definite abnormal neurologic signs, probably referable to a direct laceration of the adjacent portion of the brain. They will be considered in greater detail in the section on "follow-up."

A consideration of these ten cases sheds little light on why there should be infection in some and not in others. In some instances, a prompt operation failed to stave off the suppurative meningitis, while in others a delay of several days did not seem to cause any untoward effects. In addition, two compound fractures were described in the series of cases in which the patients recovered under expectant treatment. In general, the more the bone was broken and the more foreign material that was present in the wound, the greater was the likelihood of septic infection; some patients, however, apparently showed surprising resistance to severe injuries of this type.

(*b*) Increase of Intracranial Pressure: Twenty-three patients were operated on for the relief of intracranial pressure. Of these, twenty died and three recovered. The craniotomy in this type of lesion was not so effective as in cases in which depressed and compound fractures were the chief indications for operation.

Fatal cases. Here the points of emphasis were the general clinical picture which the patients presented in the hospital, the means by which the fracture was located and the different factors which encouraged the surgeon to try the craniotomy and to select the side of the head on which to operate.

It is worthy of note that twelve patients were admitted in deep coma and apparently in a serious condition. One of these gave a history of a lucid interval and relapse into coma a second time before he entered the hospital unconscious.

Four patients admitted in a conscious condition while in the ward rapidly became unconscious, thus demonstrating the onset of cerebral compression. Four patients were admitted in a partially stuporous condition and gradually sank deeper and deeper into the final moribund state.

The patients were operated on as follows: on the day of admission, 13; on the first day after admission, 1; on the second day after admission, 2; on the third day after admission, 1; on the sixth day after admission, 3.

As a general rule, the patients displaying the most acute symptoms were operated on as soon as possible, while others were trephined at a later date because they did not display frank signs of intracranial tension until just before the craniotomy was performed.

The external signs of injury helped to indicate, with a fair degree of accuracy, the point at which the violence was applied, but there were a few errors. An obvious laceration in one case was entirely distinct from the point of fracture, and this was explained by the fact that the head could be easily injured twice in the same casualty. However, the craniotomy site was selected on other grounds, and this furnished a check against a mistake.

The signs were: lacerations, 6; hematomas, 7; hematomas which increased in size, 1; hematomas and bleeding ear canal, 1; abrasions and bleeding ear canal, 2; bleeding ear canal, 1; no signs externally (fracture located by the roentgen ray), 1.

The presence of intracranial pressure was recognized in various ways—in many instances by a slow pulse, in some by high blood pressure, in others by pressure in the spinal fluid, in one case by increase in the size of the hematoma of the scalp, in another by profuse bleeding from a compound fracture and in four by the lapse of a conscious patient into coma while in the ward. The reliability of these various methods has already been discussed.

The neurologic signs were especially valuable in locating the site of the compression, though at times they led the operator into mistakes. The most convenient way to consider them is in connection with the complication which caused the death of the patient.

Unilateral epidural hemorrhages were demonstrated in eight instances. Two of the cases showed merely severe intracranial pressure, but not any definite neurologic signs. The other six, however, gave the following indications that the cerebral cortex was involved on one side:

Lateral linear fracture on the right side.
Epidural clot on the right side.
Deep tendon reflexes on the left more exaggerated than on the right.
Ankle clonus on the left more persistent than on the right.
The right pupil larger than the left.

Lateral linear fracture on the right side.
Epidural clot on the right side.
The right arm more spastic than the left.
Weakness of the left facial muscles.
Pupils not mentioned.

Lateral composite fracture on the right side.
Epidural clot on the right side.
Paralysis of the right facial muscles.
Extremities on the left more spastic.
The right pupil larger than the left.

Lateral linear fracture on the left side.
Epidural clot on the left side.
Paralysis of the right facial muscles.
Flaccid paralysis of the right arm and leg.
The right pupil larger than the left.

Lateral linear fracture on the right side.
Epidural clot on the right side.
Flaccid paralysis of the left arm and leg.
The right pupil larger than the left.

Lateral linear fracture on the right side.
Epidural clot on the right side.
Pressure on hematoma in the right mastoid region induced a clonic convulsion in both upper extremities.
The right pupil larger than the left. This, with a hazy eyeground on the right side, apparently determined the site of the craniotomy.

Apparently the pressure of the extradural hemorrhage over the motor area was the cause for the different neurologic reactions. In four instances the extremity muscles on the side opposite to that of the hemorrhage were involved in some way. One patient for some reason showed a paralysis of the arm on the same side as the epidural clot. The sixth case showed clonic convulsions in the upper extremities, which were induced by pressure over the right mastoid. In every instance the craniotomy successfully demonstrated the fracture and the hemorrhage.

The pupil was dilated on the side of the lesion in five cases, but was dilated on the opposite side in one case. The facial muscles reacted capriciously and were useful only as confirmatory phenomena.

Unilateral subdural hemorrhage was described in six cases. One man had every indication of severe intracranial pressure, but distinctive nervous phenomena were not present. The other five patients, however, showed some of the following definite reactions:

Causes of the Cerebral Compression	*Neurologic Signs*
Lateral composite fracture on right side running across vertex. Left subdural hemorrhage from contre-coup laceration.	Bilateral ankle clonus and Babinski sign more pronounced on the right side. Deviation of tongue to the left side. Pupils dilated but equal.
Lateral composite fracture on the right side. Right subdural hemorrhage from direct laceration.	Weakness of left arm and left side of face, manifest on second day. Deep tendon reflexes and ankle clonus increased on left side. Pupils slow to react but equal.
Posterior linear fracture to foramen magnum. Subdural hemorrhage on left from contre-coup laceration.	Clonic convulsions, right arm and leg, periodic. Later right arm became spastic and right leg flaccid. Paralysis of left facial muscles. Head turned to right, eyes to left.
Lateral linear fracture on right side. Subdural hemorrhage on left from contre-coup laceration. Bilateral craniotomies were performed in both parietal regions.	At first, neck was rigid and left facial muscles showed clonus. Periodic convulsions later. Clonic contraction of neck muscles on right and right arm. Tonic spasm of left arm. Legs—tonic spasm in extension. Pupils equal.
Lateral linear fracture on right side. Large right subdural hemorrhage from contre-coup laceration. Moderate sized epidural clot on left side.	All reflexes exaggerated on left side. Left arm and leg spastic. Right arm and leg flaccid. Right pupil larger than left.

As noted in the foregoing, in three cases, abnormal reflexes, weakness of muscle groups, clonic convulsions and tonic contractures were present on the side opposite to that where the subdural hemorrhage was located. In another instance, the convulsions and the nervous phenomena were striking but not definite, so that bilateral craniotomies were performed. In another, the pupillary reactions and spastic paralyses in the muscles of the left extremity suggested a hemorrhage on the right, but the flaccid paralysis in the right extremities indicated lesions elsewhere. As a matter of interest, there was an epidural clot of moderate size in the left temporal region, which possibly explained the latter.

In all of these cases the operative field was selected with due regard to the signs, and the source of trouble was located successfully. The pressure of the hemorrhage probably caused the abnormal neurologic conditions, although in some instances lacerations of the brain undoubtedly had an effect.

Bilateral subdural hemorrhages were present in three cases from direct and contre-coup cerebral lacerations. In one man, with a lateral linear fracture on the right side, clonic convulsive seizures were noted in the right arm and face, while the left side of the body showed almost complete spastic hemiplegia. Another patient, with the fracture on the right side, showed spasticity in both arms and legs, especially on the

right, with the development of a right patellar and ankle clonus. Still another, with a left lateral fracture, showed a flaccid paralysis of the left arm and leg and a spastic right arm and leg. The fractures in all these cases were in the parietal region, and the operation was performed near the sites of fracture.

Two patients died of a suppurative meningitis. In one instance, the craniotomy was performed in the left parietal region because a definite paralysis was present on the right side of the body and the right pupil was larger than the left. There were also definite signs of increased intracranial tension. The operation disclosed a lateral linear fracture and a direct laceration of the brain cortex. The patient later developed the meningeal inflammation which was demonstrated by lumbar puncture. Necropsy was not performed, and the cause of the meningitis could not be ascertained.

The other patient admitted showed few symptoms besides bleeding from the left ear canal. Two days later the discharge from the ear became purulent; he also developed a generalized convulsion, a stiff neck and a bilateral Kernig's sign. Indications of increased intracranial pressure appeared, which encouraged the surgeon to attempt a craniotomy in the right parietal region on the sixth day. Nothing was found at operation, but a marked cerebral hernia developed in the operative field. The patient died shortly after the operation, and necropsy disclosed a lateral linear fracture of the left middle fossa involving the middle ear, with a severe otitis media, an abscess of the brain in the adjacent portion of the left temporal lobe and a suppurative leptomeningitis. It is probable that the abscess of the brain was responsible for the symptoms of intracranial pressure.

(*b*) Recovery after craniotomy: Three patients gave distinct signs of intracranial pressure but recovered after craniotomy.

The impression was gained from the histories that all three when admitted showed only a moderately severe degree of traumatic shock.

In one instance (case 8), the fracture was located in the right temporal region by the roentgenogram and the localized headache. Definite signs of intracranial pressure on the third day of the patient's stay in the hospital, together with an engorged eyeground and an abnormal reflex on the right side, seemed to indicate a right temporal craniotomy. When this was performed, an epidural clot was found and evacuated.

In a second case (case 9), the fracture was compound in the left parietal region, while a developing right hemiplegia indicated that the complication was present near the fracture. The spinal fluid was bloody and under tension, indicating increased cerebral compression. A left parietal craniotomy, performed a few hours after the injury, disclosed an epidural hemorrhage, which was controlled.

The third case (case 10), showed bleeding from the right ear, signs of intracranial pressure, according to the blood pressure and pulse, and the deep reflexes on the left were greater than those on the right. A right subtemporal craniotomy was performed a few hours after admission, and blood and brain tissue were evacuated from the epidural and subdural spaces. The lesion was probably a laceration of the brain on the side of the fracture complicated by a subdural hemorrhage.

These cases contrasted rather sharply with the fatal ones, in which the general condition of the patient on admission, with a few exceptions, was described as serious, the degree of intracranial pressure was often much more marked, and the neurologic signs were usually more pronounced. The histories indicated that, on the whole, the intensity of the intracranial complication was much less in the patients that recovered. It was also obvious that the craniotomy was clearly indicated in all three instances, for without it death would have been inevitable.

OPERATION FOR COMPOUND FRACTURE RECOVERY

CASE 1.—I. B., a white man, aged 26, was struck by an automobile. He was conscious on admission to the hospital. A compound fracture was found in the anterior portion of the left parietal region. The left reflexes were hyperactive. Craniotomy was performed on the day of admission. The external wound was excised, and the bone was removed by rongeur forceps. The dura was intact and was not opened, but a needle was inserted, and clear cerebrospinal fluid was obtained. The length of the stay in the hospital was twenty-five days.

One year later the patient had a slight palpable pulsation at the site of the craniotomy; the left pupil was slightly larger than the right, and he showed a slight Romberg sign.

CASE 2.—J. T., a white man, aged 28, fell down a hatchway. He was conscious and rational on admission. A compound fracture involving the frontal sinus was found on the forehead. Craniotomy was performed eight days later. Recovery was uneventful; the patient was in the hospital ten days.

When this patient was seen later, a bony deformity was present at the operative site, but the patient did not have any other symptoms.

CASE 3.—J. McK., a white man, aged 55, was struck on the head by a cuspidor. On admission, he was alcoholic and stuporous, but he was easily aroused. In the left parietal region, a comminuted compound fracture containing hair was found. The patient showed a pure motor aphasia, that is, he could understand, but could not speak distinctly. Craniotomy was performed on the day of admission. After a right angle incision exposing the wound, bone fragments were removed by rongeur forceps, and a bleeding branch of the middle meningeal artery was ligated. The epidural clot was removed. A rent 2 cm. in length was found in the dura and closed. On discharge, twenty-eight days later, the patient was still a little aphasic, as he had difficulty in speaking, although he could read and write. He was also slightly dizzy. There was a sinus in the field of operation which still discharged.

Three months later, he complained of dizziness and was unable to pronounce words well. The sinus in the wound still discharged fluid. He did not report subsequently.

CASE 4.—L. D., a white man, aged 30, fell two stories to a sidewalk while cleaning windows. When admitted to the hospital he was unconscious. A compound fracture was present in the right parietal region and a hematoma of the left vault. On the second day, a craniotomy was performed in the right parietal region; a broken bone was removed, but the dura was left intact. The patient became semicomatose and had a slow pulse rate. The hematoma of the left vault increased in size. On the third day, a second craniotomy was performed over the left temporal region. A stellate fissure fracture was found with fresh blood oozing through the fracture line. A large area of bone was removed by rongeur forceps, and an epidural clot was removed. The dura was incised, and from 20 to 30 cc. of blood was evacuated. Recovery was slow. The patient was discharged at the end of forty days with injuries healed.

Six months later, the patient complained of headache and stuffiness in the ears. Mentally, he was slow and irritable. The skull defects did not bulge.

CASE 5.—W. McI., a white boy, aged 14, fell two stories. He was conscious on admission to the hospital, but irrational and irritable. A compound fracture was present in the left supra-orbital region; there was a dislocation of the left elbow and a Colles fracture of the left wrist; the neck was rigid. The spinal fluid was blood tinged; the temperature was 103.8 C. On the fifth day, a craniotomy was performed in the left supra-orbital region, and the fractured bone was removed. A torn dura was discovered and an extrusion of degenerated brain tissue, which was removed; drains were inserted in the wound. After a stormy convalescence, during which the patient had a right basal pneumonia and an infected wound at the operative site, the condition cleared up. The injuries of the left arm healed, and the patient was discharged at the end of two months and eight days, permanently blind in the left eye, except for light perception, and with a bone defect on the left side of the forehead.

Two years later, the patient returned to the hospital, and a cartilage transplant from the ribs was put in in order to correct depression at the site of the old operation. This operation was successful.

Subsequent History: When the patient was 19 years old, he accidentally fell down an elevator shaft and sustained a fracture of the skull in the right frontal region, after which he developed an acute suppurative meningitis and died. Necropsy disclosed the foregoing conditions and the old healed craniotomy in the left frontal region.

CASE 6.—F. O. P., a white boy, aged 15, was shot in the forehead. He was conscious, rational and in good physical condition on admission to the hospital. There was an operative scar on the right side of the forehead, which was almost healed. Roentgen-ray examination showed a bullet in two pieces in the outer table of the skull and a small fracture there. On the fourteenth day after admission, the bullet was removed by a craniotomy on the right side of the forehead, involving both tables. A cartilage transplant from the seventh and eighth ribs on the right side was put in in order to remedy the defect. A small fragment of bullet was left in the scalp. The patient recovered completely and was discharged after twenty-nine days.

No sequelae were found.

CASE 7.—T. S., a white man, aged 44, was hit by a falling plank. He was conscious when admitted to the hospital, but in a state of shock. A large, bulging hematoma was present in the scalp, just to the left of the median line of the vault, with a laceration in the posterior parietal region. The blood pressure was low—systolic 75, diastolic 55. Convulsive jerking of the right leg was present, and

later this extremity became weak. Roentgen-ray examination disclosed a fracture of the left parietal bone, with branches to the frontal region and to the occiput, near the leg area of the rolandic fissure. A decompression operation was performed on the fifth day in the left parietal region near the laceration. The depressed fracture was reduced by rongeur, and a cartilage graft from the ninth costal cartilage was inserted in the defect. Motion of the right leg slowly returned, although the patient walked with a slightly spastic gait. There was no defect in the skull. The patient was discharged twenty-six days after admission.

One year after operation, no untoward symptoms persisted, with the exception of a slight Romberg sign, and the right knee reflex was a little exaggerated.

INTRACRANIAL PRESSURE

CASE 8.—A. D., a man, aged 41, slipped and fell, striking the right side of his head. He was unconscious for one hour. He vomited about ten hours later and had a hemorrhage from the nose, mouth and right ear. Pain then developed in the right temporal region, and on account of this symptom, he entered the hospital three days later. On admission, he was conscious, and any external sign of injury could not be seen, but a definite point of tenderness was found 2 cm. above the right temperomandibular region. Roentgen-ray examination showed a linear fracture in this region. The right pupil was somewhat larger than the left, and both reacted sluggishly to light and accommodation. Blood was present in the spinal fluid, but the pressure was not increased. Three days after admission, an intravenous administration of 50 cc. of a 3 per cent sodium chloride solution was given. The pulse dropped from 77 to 54 beats a minute; the right eyeground showed dilated veins and narrowed arteries, but the disk itself was normal; there was a positive Oppenheim sign on the right, and persistent headache developed. A right temporal craniotomy was then performed, and a stellate fracture of the right temporal bone was found, one fragment being depressed. The bone was removed with rongeur. A thick epidural clot was removed with a curet, and the posterior branch of the middle meningeal was found lacerated near the bifurcation; this was ligated, and the oozing from the dura was controlled by a postage stamp graft of muscle. Recovery was uneventful. The patient was discharged at the end of twenty-eight days.

There were no sequelae.

CASE 9.—W. W., a negro, aged 27, was hit by a falling brick. He was admitted in a semicomatose condition. A compound depressed fracture was present in the left parietal region. A hematoma of the right eyelid was present. A right hemiplegia rapidly developed, except in the fingers, accompanied by a pulse rate of 92 beats a minute and a blood pressure of 110 systolic and 50 diastolic. The spinal fluid was bloody and under pressure. A left parietal craniotomy was performed on the day of admission. A depressed fracture was found, with hair and débris in the wound. The bone was removed by rongeur forceps, leaving a defect 6 by 5 cm. well beyond the soiled area. A thick layer of epidural clot was found and removed by curet. Tears of a branch of the middle meningeal artery and of the superior longitudinal sinus were found, and the bleeding points were controlled by application of aponeurotic grafts. The dura appeared normal and was not opened. There was gradual recovery of movement on the right side; the arm movements came back first; the right leg improved slowly and, even on the day of discharge, the patient had a weak ankle and a steppage gait. He was discharged twenty-four days after injury.

The patient was seen over a period of two years. Slow improvement took place, and he regained power in the right leg gradually. He was able to do heavy work, but there was some limitation of motion in the toes. The bone defect did not cause any trouble.

CASE 10.—V. N., a man, aged 25, was injured in collision between a truck and a street car. He was admitted to the hospital in a rational condition, but was drowsy and irritable. There was bleeding from the right ear canal, and the ear was split in a portion of the tragus. The spinal fluid was bloody. A few hours later, he became drowsier and more irritable. The deep reflexes were exaggerated, the left more so than the right. Blood pressure was 160 systolic and 120 diastolic. The pulse rate was slow, but full and bounding. A right subtemporal craniotomy was performed a few hours after admission, and an extensive egg-shell fracture was found in the temporal fossa. The bone was removed by rongeur forceps. A small amount of clotted blood and brain tissue was found external to the dura. As small spicules of bone had pierced the dura, a crucial incision was made in the dura. The brain bulged slightly and pulsated. Recovery was not eventful; pulsation at site of operation subsided, and the patient was discharged after twenty-six days in the hospital.

A month later, the patient complained that he was easily irritated by loud noises, had a headache and was dizzy. Three years later, he was reported as free from objective symptoms, but was unable to work in the city because he could not endure the noise.

CRANIOTOMY FOR THE RELIEF OF A DISABLING PARALYSIS

CASE 11.—R. C., a white woman, aged 36, was struck by an automobile and knocked against an elevated pillar. She was at another hospital for eleven days and then transferred to Bellevue. Shortly after admission, she showed a laceration of the scalp in the left temporoparietal region. There was a left facial paralysis of the upper motor neuron type. Roentgen-ray examination showed a stellate fracture of the left parietal region. On admission to Bellevue, the patient was semistuporous, restless and moved all extremities with the exception of her left arm, which was rigidly flexed on the chest. There was a left lower facial paralysis. The tongue protruded to the left. The symptoms were diagnosed as a possible injury to the right motor area. A craniotomy was performed in the right parietal region over the motor area nineteen days after the injury. A bone flap was turned down, the motor area exposed, a needle inserted in the arm area and some old blood obtained from the brain substance. Nothing further was done. The paralysis of the arm improved gradually, and the patient left the hospital on the fifty-third day after the injury.

At the end of two years, the patient did not show any particular symptoms except a slight spasticity of the left leg, but this was not troublesome. Otherwise she was in good condition.

The eleven cases in this series showed a variety of after-effects. One boy recovered and remained without symptoms (case 6). Two other patients also remained without symptoms, with the exception of the bone defect at the craniotomy site (cases 2 and 8). Three patients recovered without any other symptoms except some vague neurologic signs (cases 1, 4 and 7). One man (case 10) developed a marked psychosis, so that he was unable to work.

Four patients, however, showed definite sequelae, which in all probability were referable to some lesion of the brain or some other complication of the fracture. These sequelae are shown in table 21.

In cases 3, 9 and 11, the muscular disability never disappeared entirely but improved as time went on. This suggests a brain injury in the cortex as the cause of the symptoms.

The craniotomy wound in the eleven cases caused scarcely any trouble, except in two instances. One man showed a pulsation at the site of the defect (case 1). In others, a deformity was left on the skull, notably a depression near the decompression window. One boy (case 5) actually returned to the hospital to have the defect successfully corrected by a cartilage transplant from the rib. Two others (cases 6 and 7) had the skull defect similarly reinforced at the operation, and the result was uniformly successful.

TABLE 21.—*After-Effects of a Compound Fracture and the Probable Cause*

	Disability	Probable Cause
Case 5	Total blindness of one eye, permanent	Cause unknown, but probably due to tear of optic nerve or injury of eyeball
Case 3	Persistent aphasia; motor speech	Probable injury of brain near island of Reil
Case 9	Loss of power in right leg which, however, improved	Probable injury to brain in motor area in left cerebral hemisphere
Case 11	Slight spasticity of left leg, not troublesome	Definite injury of brain in motor area in right cerebral hemisphere

RECOVERY UNDER EXPECTANT TREATMENT

Many patients with injury of the head were admitted to the hospital and recovered under expectant treatment, but it was questionable whether all of these had fractures of the skull. Unless a definite crack in the bone could be discovered by direct observation or by roentgen-ray examination, the case was eliminated from the series.

The total number of cases was twenty-seven. Two fractures were seen or felt through a laceration of the scalp in the frontal and right parietal regions, respectively. Another fracture of the frontal region was diagnosed by the presence of comminuted nasal bones, hematoma of the eyelids and a subcutaneous emphysema around the frontal region. The remaining fractures, however, were demonstrated only by the roentgen-ray examination; there were ten in the right parietal region, nine in the left parietal and five in the occipital region.

The preponderance of parietal fractures in the series was, perhaps, due to the fact that the roentgenogram shows cracks more readily in the lateral part of the skull than elsewhere, probably because the photographic field is easier to penetrate in this part than in the other regions.

As a means of diagnosis, the roentgen ray is valuable in some cases, but the possibility of error cannot be excluded. Many grooves occur in the skull normally, and it is not impossible that some of these may throw a shadow similar to a skull fracture. However, in this series positive diagnosis by the roentgen ray was accepted as proof of fracture.

The age and sex incidence are shown in table 22.

On admission to the hospital the symptoms were variable, but were generally those of a moderately severe cerebral concussion. Three patients were unconscious but recovered their senses a few hours afterward. The other twenty-four were conscious on admission to the hospital and more or less oriented; they were semistuporous, irritable, complained of headache, had vertigo, vomited, etc. Most of these patients gave a history of a period of coma immediately after the accident; in other instances, their condition after the accident was in doubt. One man said definitely that he was not unconscious.

TABLE 22.—*Cases that Recovered Under Expectant Treatment*

Age Incidence			
	No. of Cases		No. of Cases
10 to 20 years	5	40 to 50 years	4
20 to 30 years	4	50 to 60 years	5
30 to 40 years	8	60 to 70 years	1
Sex Incidence			
22 men		5 women	
Casualty Incidence			
Automobile accidents	6	Hit by falling object	2
Street car accidents	1	Hit by digger on coal barge	1
High falls	3	Elevator accident	1
Low falls	4	Beaten	1
Falls down stairs	3	Unknown	1
Thrown from vehicle	4		

Some of the patients improved without displaying any further symptoms, while others gave an indication that they were suffering from a mild grade of cerebral compression. This was shown in various ways—by a slow pulse rate, by increased pressure in the spinal fluid and by a variation in blood pressure from systolic 90 and diastolic 60 to systolic 150 and diastolic 70. The symptoms were never extremely severe, however. Sometimes the headache was localized, and this, together with vertigo and vomiting, was the only phenomenon recorded. None of the patients in this series showed a second lapse into unconsciousness so often described in the operative cases.

The external signs of injury were various. Lacerations of the scalp were present in twelve patients, contusions of the scalp in seven, hematomas of the eyelids in three, bleeding from the ear in five and from the nose two, vomiting of blood in three, blood in the posterior pharynx in one and subcutaneous emphysema of the frontal region in one. Five

cases did not present any sign of head injury. In the majority of instances, the external injuries gave a clue to the location of the fracture, but in some this connection was not obvious.

In two instances a sharply localized headache, once in the left parietal area and once in the occipital area, helped to indicate the probable site of the fracture, which was later confirmed by roentgen-ray examination.

The neurologic signs were indefinite. All the pupillary reflexes were normal. Examination of the eyegrounds revealed at most only a slight haziness or congested vessels. The normal reflexes were described as hyperactive, sluggish and sometimes unequal on the two sides. A few pathologic reflexes were described, such as ankle clonus, Babinski sign, positive Kernig sign, stiff neck, nystagmus, temporary facial paralysis and deviations of the tongue to one side. One woman showed a permanent foot drop, but there was reason to believe that this was caused by an injury to the peripheral nerve trunks. Most of these signs were temporary, and were not definite enough to encourage a craniotomy for the relief of the symptoms.

Examination of the spinal fluid revealed a bloody fluid in two or three tubes in eighteen cases (under pressure 10), a yellow tinged spinal fluid in 2, a spinal fluid at first bloody, then clear in 1, observations not recorded in 6. The blood in the fluid indicated that there was a subarachnoid hemorrhage of some kind which, in all probability, took its origin from an injury to the brain. The yellow tinged fluid occurred in two patients who entered the hospital several days after the trauma, and the lesion presumably was being transformed into a "plaque jaune" at the time of the lumbar puncture. One patient, as noted, showed blood in the spinal fluid on admission to the hospital, which later became clear, probably indicating complete healing of the brain injury.

The anatomic characteristics of the fracture and the intracranial lesion could only be surmised. Most of them appeared to be vault fractures which descended into the base. With the exception of one or two, it was impossible to say how many were comminuted. Two fractures were compound, but neither was infected, and it is difficult to explain why they should have healed without operation, while intracranial infection occurred in similar lesions.

Twenty-one patients ended their convalescence in from twenty to twenty-four days. Two were in the hospital only fourteen days, but had been injured several days previous to admission. Four patients remained from thirty to fifty days in the ward, but this was because they had other injuries, such as fractures of the forearm and the like. The usual term of convalescence seemed to be slightly more than three

weeks. The impression was given that the active symptoms would clear up during that time if the injuries were going to heal spontaneously.

All of these twenty-seven patients were followed up for a greater or lesser length of time after discharge from the hospital ward. Nine did not show any symptoms. The other patients, however, complained of a great variety of symptoms, some of which were transient, while others lasted as long as the follow-up observation continued. Fifteen patients complained of headache and dizziness. Three others showed symptoms of a low grade psychosis, such as apprehensive feelings, poor memory, inability to concentrate, and the like. Undoubtedly many of these phenomena had an organic basis, but some were undoubtedly psychologic. Fractures of the skull are regarded with great trepidation by the laity, and there is a tendency to exaggerate the consequences of the injury. It was also difficult to say in any one case just what was caused by the fracture and what was the result of some other constitutional disability.

Six patients developed deafness in one ear, four on the right and two on the left side. There was reason to believe that the disability did not antedate the injury and was caused by the fracture line running through the ear structures in the middle fossa. Four showed bleeding from the auditory meatus on admission to the hospital, and five showed fractures involving the middle fossa in the ear region. The lesion which produced the deafness was never ascertained. Otoscopic examination of the ear drums was recorded in three cases, but only one patient showed a definite injury of the drum on the deaf side (see protocols).

In this connection, it was worth noting that two patients who showed bleeding from the external auditory meatus on admission did not subsequently complain of deafness in that ear. Fractures of the middle fossa not infrequently passed over the roof of the external ear, avoiding the deeper ear structures, but at the same time causing an aural hemorrhage by laceration of the mucous membrane of the external canal. This lesion would not necessarily cause deafness. One case in the list showed a temporary facial paralysis which, however, cleared up in six months after the patient left the hospital. The origin of the paralysis was not obvious (case 15).

One patient who was followed up complained of a greatly impaired sense of smell, which gradually improved as time passed. The history and clinical course in this case suggested that the disability was, in all probability, caused by lacerations on the under surface of both frontal lobes which involved the olfactory bulbs to a greater or lesser degree. Many necropsies showed numerous contre-coup lacerations of the orbital convolutions and the olfactory bulbs, and it was more than probable that this was the lesion in case 18.

CASES OF DEAFNESS

CASE 12.—M. N., a white man, aged 40, fell down stairs while intoxicated. He was stuporous on admission, but was able to talk. A V-shaped laceration was present in the right parietal region, and there was bleeding from the right ear canal. Roentgen-ray examination showed a fissure fracture of the right temporal bone, running perpendicularly into the right ear canal. The fluid obtained by lumbar puncture was uniformly bloody. When the acute symptoms subsided, it was found that the patient had persistent deafness in the right ear and a feeling of vertigo. Deafness persisted during the length of the patient's stay in the hospital. On otoscopic examination, the left ear drum was normal and the right ear drum showed a little blood clot attached to the surface.

After one year and six months, there was complete deafness in the right ear and persistent vertigo.

CASE 13.—F. S., a white man, aged 18, fell from a fire escape. He was admitted to the hospital in coma, but recovered his senses in a few hours. There was an abrasion on his head in the frontal region, bleeding from the nose and vomiting of blood, but no bleeding from the ears. The ear drums were white. The spinal fluid was bloody and under pressure. All of the reflexes were exaggerated, with ankle clonus and a bilateral Babinski sign. The pupillary reactions were normal, but nystagmus was present in each eye. Roentgen-ray examination revealed a fracture in the right temporal bone, running to the right auditory meatus. The vertigo and headache on the right side persisted, and there was complete deafness in the right ear. The patient was discharged at the end of twenty-one days.

For three years the patient showed complete deafness in the right ear and for a long time daily headaches.

CASE 14.—J. D., a white man, aged 23, fell 15 feet, landing on his head. He was admitted to the hospital in a semiconscious condition which persisted for ten days. There was a laceration in the right parietal region, bleeding from the right ear and vomiting of food mixed with blood clot. Roentgen-ray examination showed a fracture in the right temporal region. The spinal fluid was bloody and under pressure. The pulse rate was slow, about 50 beats a minute, and the blood pressure was high for several days—systolic 150 and diastolic 70. The symptoms gradually improved, and the patient was discharged at the end of twenty-two days.

Three months after discharge, the patient complained of deafness in the right ear with a tendency to stagger to the right At the end of two years, the deafness persisted, but he did not stagger unless he was tired.

CASE 15.—F. Y., a white man, age not stated, was thrown from a truck by a kicking horse. He was conscious on admission to the hospital, but apparently quite ill. There was a small hematoma over the right eye and bleeding from the right ear. The spinal fluid was blood tinged. There was complete facial paralysis, with distinct nystagmus to the left and deviation of the tongue slightly to the left. The pupillary reflexes and tendon reflexes were normal. Roentgen-ray examination showed a fracture in the right temporal bone. The patient complained of persistent deafness in the right ear and of a headache on the right side. The facial palsy also persisted. He was discharged at the end of twenty days with these symptoms persisting.

The dizziness, headache and deafness persisted, but the facial paralysis cleared up in about six months. The deafness was unchanged at the end of thirteen months, but the headache and dizziness were much less intense.

CASE 16.—J. O., a white woman, aged 25, was knocked down by a taxi. She was admitted in a stuporous condition, with tender spot just behind and above the left mastoid. She complained of severe occipital headache and ringing in the left ear. The spinal fluid was bloody on admission, but flowed out clear seventeen days later. At first, she showed definite nystagmus, an active patellar tendon reflex on the right and absent on the left. Roentgen-ray examination disclosed a fissure fracture of the right side of the occiput running to the foramen magnum and another fracture of the right border of the foramen magnum. She was discharged on the twenty-fourth day, still complaining of deafness and buzzing in the left ear.

One year later, she showed persistent deafness in the left ear, but no other symptoms.

CASE 17.—C. B., a white man, aged 27, was injured in an elevator accident. He was admitted in a semistuporous condition with pain in the back of his head. Bleeding from the right ear was present. The pupillary and tendon reflexes were normal. The spinal fluid was bloody but under normal pressure. Roentgen-ray examination disclosed a fracture in the right middle fossa, running into the right temporal and parietal bones of the vault. He complained of deafness in the right ear, which persisted to the day of discharge—twenty-four days later.

After two years, deafness persisted in the right ear, and the patient also had a low grade psychosis.

LOSS OF SENSE OF SMELL

CASE 18.—H. R., a white man, aged 43, was struck by a taxi. He was disorientated and drowsy on admission to the hospital, but he knew his name and address. He complained of headache and dizziness, especially on motion. External injuries were not noted. The pupillary reactions were normal, and the tendon reflexes were slightly hyperactive. The spinal fluid was bloody and under increased pressure. There was slight cloudiness in the right eyeground; the left was normal. The day after admission, the patient complained of right frontal headache, which persisted for three days. A slight ecchymosis developed in the right eyelid. Roentgen-ray examination showed a fracture in the right temporo-occipital region, extending about 1½ inches upward from a point 2½ inches behind the right auditory meatus. For a few days, his pulse rate was slow—about 68 to 80—with a blood pressure of 130 systolic and 80 diastolic. All symptoms finally cleared up, and on the twenty-fourth day the patient was discharged.

For a few months, he suffered from dizziness on sudden motion of the head and throbbing in the left ear. He also found that he had lost all sense of smell; at first, this was complete in the left nostril but only reduced in the right. This persisted, but gradually he was able to recognize odors. He found that he often noticed smells that were not present, such as the odor of creosote. Most odors were disagreeable to him. This did not have any relation to his physical condition, as he was well otherwise.

COMMENT

The sixty-one cases considered showed a preponderance of fractures in the lateral part of the skull, with relatively few located anteriorly and posteriorly. They were found as follows: lateral fractures, 48; anterior fractures, 7; posterior fractures, 6. The reason for this disproportion was probably that the lateral variety was more easily discovered by the roentgen ray than the other types, and also that this variety was accompanied by such characteristic external lesions as large hemor-

rhages of the scalp or bleeding from the external ear canal. In addition, lateral fractures usually involved the motor cortex in various ways and thus produced striking neurologic signs, while anterior and posterior fractures, even when severe, tended to involve the "silent" areas of the brain and only occasionally elicited a definite neurologic phenomenon.

In general, with the exception of such outspoken indications as compound or depressed fractures, patients with the anterior and posterior group of lesions did not require operation in the normal course of events. They seemed either to cause complications which were almost inevitably fatal or else to be accompanied by such slight intracranial lesions that the patient recovered spontaneously.

Lateral fractures displayed these same characteristics to a certain extent, but at the same time produced a large number of intracranial injuries which were amenable to a craniotomy, but which invariably proved fatal if left alone. Accordingly, lateral fractures formed the vast majority of those in patients subjected to craniotomy.

SUMMARY

1. A number of clinical records were collected, and the reaction of the patient to the fracture of the skull during life was studied.

2. Many patients were admitted to the hospital in a moribund condition or lingered for several days in the hospital, finally dying of suppurative meningitis, pneumonia or sepsis. They were cases not suitable for operation because of the patients' poor physical condition and because of the absence of signs which indicated a specific condition that would be alleviated by craniotomy.

3. Sixty-one cases were studied in some detail. They were arbitrarily separated into the following categories: (1) patients on whom a craniotomy had been performed, thirty-four; twenty-three of these patients died and eleven recovered; (2) patients recovering under expectant treatment, twenty-seven.

4. Patients treated by craniotomy were subjected to rather rigid physical examination before the procedure was attempted. It was first necessary to determine the probable location of the skull fracture by a careful consideration of the history of the case, the observations elicited on clinical examination, the appearance of the spinal fluid, the types of external injuries present, and by roentgen-ray examination.

5. Then it was necessary to determine whether there were signs of an intracranial condition that made craniotomy advisable. In the thirty-four patients operated on, three indications for operations were discovered: (*a*) compound or depressed fractures of the skull (three patients died and seven recovered); (*b*) the presence of definite intracranial tension, especially if the lesion causing the trouble could be definitely localized

by neurologic examination (twenty patients died and three recovered), and (*c*) the presence of a disabling paralysis on one side (one patient recovered).

6. The twenty-seven patients who recovered under expectant treatment were all practically over the acute effects of the injury in from twenty to twenty-four days after the trauma. As a rule, they displayed only moderately severe symptoms of cerebral concussion and compression. The fracture was located by the roentgen-ray examination in twenty-four cases, through a compound fracture in two instances, and by subcutaneous emphysema of the frontal region in one. The neurologic examination and other signs were never definite enough to justify a craniotomy.

7. The patients who recovered were followed up for variable periods until it was felt that they had definitely recovered from the results of the fracture or until they disappeared from observation.

(*a*) The eleven patients who recovered under operation showed a variety of phenomena. Some were practically without after-effects or, at the most, displayed indefinite symptoms, such as headache or vertigo. One man developed a low grade psychosis. Four patients, however, had some definite disability, such as the loss of sight in one eye in one case, motor aphasia in another case and definite weakness in one leg in two instances. The motor disabilities improved but did not disappear entirely. (*b*) Most of the twenty-seven patients recovering under expectant treatment did not show any severe after-effects, except vague symptoms of headache, dizziness, and the like. A few, however, had minor disabilities which could be attributed to definite lesions. Six were permanently deaf on one side, while one man showed a disturbance of the sense of smell.

LOBAR ATELECTASIS IN CHRONIC PULMONARY SUPPURATION *

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Observations on massive collapse of the lung and on other clinical conditions presenting extensive atelectasis enable us to offer a more complete description of the characteristic roentgen-ray shadow cast by a single atelectatic lower lobe, and to call attention to its association with certain cases of chronic pulmonary suppuration. Lobar atelectasis has been identified by Manges [1] through roentgen-ray observations in cases of obstruction of the air passages by foreign bodies. It has been observed in postoperative massive atelectasis of the lung by Jackson and Lee [2] and by Churchill.[3]

ROENTGEN-RAY OBSERVATIONS IN LOBAR ATELECTASIS

A deflated lobe of the lung is commonly pictured as a small puckered mass retracting to the hilum by means of a bellows-like shortening of the bronchi. This is what happens when the deflation occurs as the result of pneumothorax or pleural effusion; but when it is produced by bronchial obstruction in a chest with an intact pleural cavity, a different picture is presented.

The shape which a deflated or atelectatic lobe takes in the latter case is molded to conform to the adjacent lobes and structures and is determined by its inherent elasticity. The characteristic outline of an atelectatic lobe has already been demonstrated [4] by a roentgenogram of an autopsy specimen after its removal from the body. The shape assumed by a collapsed lower lobe is roughly that of a right-angled triangle, the base of which rests on the diaphragm. The apex is held at the hilum by bronchial attachments, and the hypotenuse shows as a sharply defined border which extends from the hilum to the diaphragm near the costophrenic angle.

Although the cases of postoperative massive atelectasis usually described show involvement of an entire lung, certain cases with a

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* From the Massachusetts General Hospital.

1. Manges, Willis F.: Am. J. Roentgenol. **10**:517, 1924.
2. Jackson, C., and Lee, Walter E.: Ann. Surg. **82**:364, 1925.
3. Churchill, Edward D.: Pulmonary atelectasis; with special reference to massive collapse of lung, Arch. Surg. **11**:489, 1925.
4. Churchill, Edward D. (footnote 3, fig. 9).

lobar distribution of the collapse have been observed. The roentgenograms of two such cases have been reproduced in a previous paper by one of us. In the first case there described,[5] the lobar collapse persisted as a residual condition after the other lobes were reinflated; in the second case,[6] the collapse when first observed was confined to the lower lobe.

It has been shown by Jackson and his co-workers that although the lodgment of a foreign body in a bronchus may produce hyperinflation under certain circumstances, complete obstruction causes deflation of the portion of the lung to which the bronchus is contributory. There

Fig. 1.—Atelectasis of the right lower lobe from the impaction of a meat bone in the bronchus.

may also be an attendant "drowning" of the lung and bronchial tree by the retained secretions. The roentgenogram reproduced in figure 1 shows a patient with a meat bone impacted in the bronchus of the right lower lobe. It shows the characteristic shadow of lobar collapse.

A tumor of the bronchus may by its growth so occlude the lumen as to produce complete atelectasis of a lobe. The roentgenogram reproduced in figure 2 is that of a patient with a primary squamous cell carcinoma obstructing the bronchus to the right lower lobe. The diagnosis was made on the basis of the history and roentgen-ray observations and confirmed by a bronchoscopic examination by Dr. D. Crosbie Greene. As in cases of massive collapse of the lung, the shadow of the collapsed

5. Churchill, Edward D. (case 1, fig. 2).
6. Churchill, Edward D. (case 2, figs. 3 and 4).

lobe obliterates the cardiophrenic angle, its upper border extending as a sharply defined line from the diaphragm to the hilum.

These observations, supported by further evidence obtained from the cases to follow, make it apparent that an atelectatic lower lobe appears in the roentgenogram as a triangular opacity which obliterates the cardiophrenic angle on the right, but is largely obscured by the shadow of the heart on the left. The apex of the triangle lies at the hilum, and the hypotenuse extends outward and downward either to merge with the shadow of the diaphragm or to meet the chest wall near the costophrenic angle.

Fig. 2.—Atelectasis of the right lower lobe from the obstruction of a primary carcinoma of the bronchus.

REPORT OF CASES

Roentgen-ray examination in the following cases of chronic broncho-pulmonary suppuration showed the shadow of a deflated lower lobe.

CASE 1.—F. S., a girl, aged 18, with an uneventful past history, became ill on Dec. 12, 1923, with chills, fever and an unproductive cough. After a few days in bed she raised a small amount of thick, greenish sputum. Two weeks later, positive cultures were obtained from her throat, and she was taken to the diphtheria ward of the Boston City Hospital. There she developed a postdiphtheritic paralysis which caused her to remain for some five weeks.

During this period she had a cough, and the amount of sputum increased to several cups a day. It gradually became foul. After three months in another hospital, she entered the Massachusetts General Hospital in June, a little more than six months after the beginning of her illness. Her condition had been stationary for four months, the chief symptom being the production of about 15 ounces of foul, purulent sputum a day.

Examination showed that she was fairly well nourished, with a foul breath and an almost constant cough. There was definite clubbing of the fingers of both hands. Over the back of the right side of the chest, there was dulness from the angle of the scapula to the base. The breath sounds were bronchial at the top of this area, but absent at the base. There was egophony from the angle of the scapula downward. Vocal and tactile fremitus were increased in this region, and râles were not evident. The sputum was negative for tubercle bacilli, and bacteriologic examination showed it to be more suggestive of bronchiectasis than of lung abscess.

Roentgen-ray examination of the chest (fig. 3) showed evidence of "consolidation at the right base involving the entire lobe." Evidence of cavity formation was lacking. The heart was displaced toward the affected side. These observa-

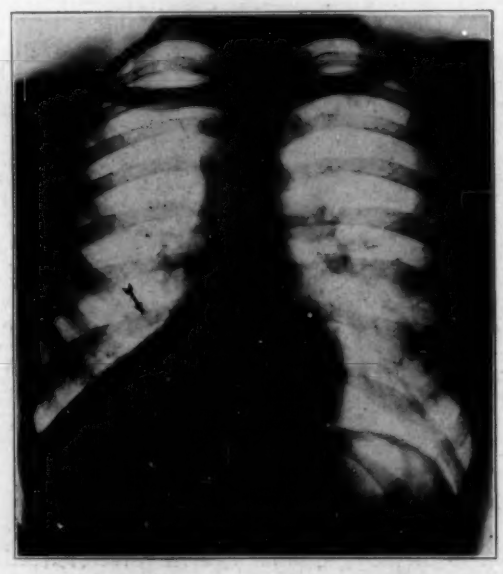

Fig. 3 (case 1).—Atelectasis of the right lower lobe with chronic pulmonary suppuration following postdiphtheritic paralysis.

tions at the time were thought to be consistent with an obstruction of the bronchus by a foreign body, pneumonic consolidation or abscess formation.

An operation was performed by Dr. Wyman Whittemore on June 19. The pleural cavity was found to be free from adhesions, with the exception of a few fibrous bands between the lower lobe and the diaphragm. The right lower lobe was small, of firm consistency and a dusky blue. The upper lobes were of normal appearance. At the first operation the lower lobe was walled off from the pleural cavity; following its removal a week later, the patient died.

Bronchial obstructions were not found in the excised lobe. The bronchi were filled with purulent exudate, and their walls were described as dilated[7] and surrounded by round cell infiltration.

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7. A statement that a bronchus is dilated should be based on a consideration of the diameter in relation to the distance from the hilum, and not in relation to the proximity of the pleural surface or of other bronchi of similar diameter when

CASE 2.—M. D., an Italian youth, aged 18, gave a history of double pneumonia at the age of 5. He had been in bed for three months during a prolonged convalescence, and since that time had been subject to colds and bronchitis, particularly in the winter. He dated his cough and sputum, however, to a serious illness following an attack by two dogs five years before coming to the hospital. He was severely bitten about the buttocks and on the body, and was confined to bed for several weeks.

He was first examined in May, 1922, when he complained of a productive cough. There were musical râles at the bases of both lungs, and physical examination revealed that the cardiohepatic angle was obliterated. In December of the same year, he contracted a severe cold attended by an increase in the cough and in

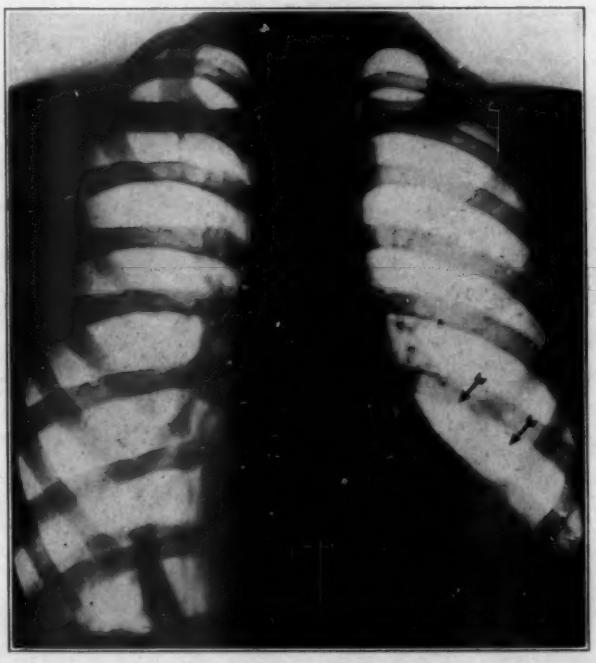

Fig. 4.—Atelectasis of the right lower lobe with chronic pulmonary suppuration (case 2). The displacement of the heart to the affected side is more marked than usual.

the amount of sputum. The latter gradually increased to a cupful a day. It became very foul and was at times streaked with blood after a severe attack of coughing.

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seen in cross-section. A cross-section through a collapsed lobe shows a cluster of large bronchi lying in close proximity to each other and to the visceral pleura. Such a picture in an inflated lobe would indicate a pathologic enlargement of their diameters, but in a collapsed lobe must be interpreted with caution. This point will be made clear by reference to figure 1 in the article referred to in footnote 3, where the bronchi in a collapsed lobe are outlined by the roentgenographic density of their cartillaginous rings. It is also important to bear this fact in mind in the interpretation of injections of iodized oil into the bronchial tree, as such a cluster of bronchi of normal size may give the impression of a large cavitation.

The patient lost weight and strength steadily during the next five months. A roentgenogram (fig. 4) showed marked dulness in the lower portion of the right part of the chest, with a sharply defined upper border extending downward and outward from the hilum to the axillary margin, obliterating the shadow of the diaphragm and the outline of the right border of the heart. This appearance was thought to be due to a chronic destructive process involving the right lower lobe and causing retraction of the heart and mediastinum to the right. It was remarked that occlusion of a bronchus might produce such a picture.

When he was admitted to the hospital in August, 1923, he was raising from 6 to 15 ounces of foul green sputum a day. On examination this showed many influenza bacilli, intracellular and extracellular; diphtheroid bacilli and a few intracellular streptococci. Repeated examinations failed to show tubercle bacilli.

Fig. 5 (case 2).—Injections of iodized oil, showing the saccular dilatation of the bronchi of the collapsed lobe in the presence of a therapeutic pneumothorax. (Injection mass traced with ink.)

Examination of the chest showed that the heart was displaced to the right. There was dulness in the lower portion of the chest with increase of fremitus and bronchial breathing. A diagnosis of bronchiectasis was made after a prolonged study to rule out tuberculosis.

Artificial pneumothorax was induced by Dr. Balboni and increased by refills until the lung was completely collapsed. As the lower lobe remained in contact with the diaphragm in its inner half, it is probable that there were some adhesions in this region. There was a reduction in the amount of sputum for a few months, but later the patient began to raise about 10 ounces a day. The amount of sputum continued the same, even when a complicating pleural transudate almost filled the right part of the chest. The fluid was aspirated from the pleural cavity many times, and unsuccessful attempts were made to reexpand the lung with view to a

radical surgical procedure. In November, 1925, Dr. Greene examined him with a bronchoscope and did not find any evidence of bronchial obstruction. An injection of iodized oil, 40 per cent, into the right lower lobe in the presence of the pneumothorax showed the saccular cavitations characteristic of bronchiectasis (fig. 5).

The pleural cavity ultimately became infected from a spontaneous fistula at the site of a needle puncture. After drainage of the empyema the patient's condition improved for a time, but he died following the first stage of a thoracoplasty.

CASE 3.—V. W., a housewife, aged 31, was operated on at another hospital in February, 1922. A curettage, suspension of the uterus, removal of an infected ovary and routine appendectomy were performed under ether anesthesia. Imme-

Fig. 6 (case 3).—Lobar atelectasis with chronic suppuration.

diately following the operation, she had a dry cough which was soon productive of thick green sputum. She lost weight as the cough continued, and experienced frequent night sweats. After two months there was a short remission, but with a cold the symptoms returned and were progressive. The sputum increased in amount and became more foul.

Eleven months after the onset of the cough, she was admitted to a sanatorium, but the diagnosis of tuberculosis was never established. She was referred to the Massachusetts General Hospital in April, 1923, fourteen months after the symptoms appeared.

When the patient was examined, dulness was found at the base of the right lung below the angle of the scapula, with diminution of the vocal and tactile fremitus and breath sounds. There were a few moist râles in this region, and egophony was noted at the angle of the scapula. There was clubbing of the fingers.

Fig. 7 (case 3).—After a partial pneumothorax. The outline of the affected lobe is thrown into relief by the surrounding air. The upper arrow denotes the border of the partially collapsed upper lobes, which show a noteworthy difference in density in contrast to the lower lobe.

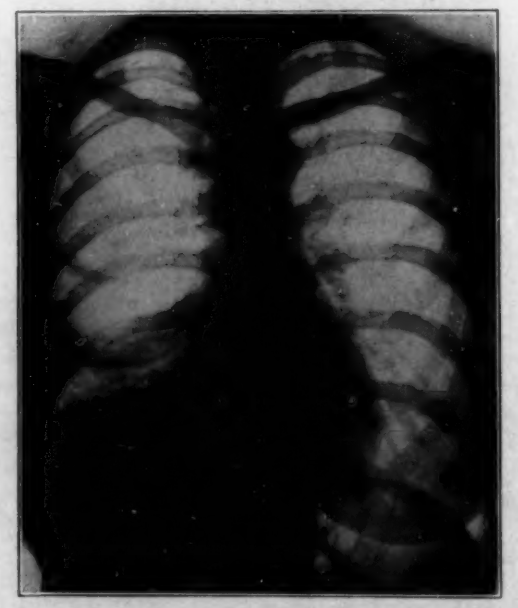

Fig. 8 (case 3).—After discontinuance of the therapeutic pneumothorax. The cardiophrenic angle is still hazy, but both the right border of the heart and the entire extent of the diaphragm are visible.

The sputum averaged 7 ounces a day, and was foul. Microscopic examination showed a profusion of organisms with many streptococci. The influenza bacillus was seen, both intracellular and free. Tubercle bacilli were not found.

Roentgen-ray examination (fig. 6) revealed an area of mottled density which obliterated the angle between the diaphragm and the heart on the right side. The level of the diaphragm was high, and its respiratory movements were limited. These observations were interpreted as those of an extensive pathologic process near the right descending bronchus confined to the lower lobe of the right lung, probably an abscess.

A portion of the ninth rib was resected under local anesthesia. The lung collapsed when the pleural cavity was opened so the incision was closed and the pneumothorax maintained by refills (fig. 7). The reduction in the amount of sputum during the following seven weeks was slight, although the refills seemed to relieve the coughing spells temporarily.

The right phrenic nerve was then sectioned, and the artificial pneumothorax was continued by Dr. Balboni for several months, after which the lung was allowed to reexpand. In August, 1924, the patient was much improved, but was still raising 1½ ounces of sputum a day. The roentgenogram after reexpansion (fig. 8) showed some residual density at the right base, but the outline of the entire right half of the diaphragm was visible, and the typical lobar distribution of the shadow had disappeared.

CASE 4.—M. K., a housewife, aged 39, had been confined to bed for five days with an attack of influenza six years before admission. At this time she coughed up a considerable quantity of sputum, but both the cough and sputum quickly disappeared without any residual symptoms except a slight increase in a chronic nasal catarrh.

On April 17, 1922, she submitted to an operation in another institution for a pelvic tumor. Two weeks previously she had had a cold in the head which had not entirely cleared up at the time of operation. She was tired and exhausted by the operation, which was attended by considerable hemorrhage. The wound subsequently became infected and discharged foul pus.

Immediately following the operation she felt "choked" by an accumulation of phlegm in her nasopharynx and throat. She was unable to expectorate because of weakness. About three days later she began to cough, raising thin watery sputum which soon became thick and green. She continued to cough, and the sputum increased in amount *but did not become foul until a month after the operation.* Any sudden change in position, such as turning in bed, induced an attack of coughing. There was never any pain suggesting pleural involvement, nor any fever.

She entered the Massachusetts General Hospital in June, 1922, a little more than two months after the operation from which she dated her pulmonary symptoms. On examination it was seen that the right half of the chest expanded less than the left. Dulness, diminished breath sounds, diminution of the whispered voice and absent tactile fremitus at the right base were present, but râles were not evident. There was slight clubbing of the fingers. Several ounces of foul, purulent sputum were raised daily, but examination failed to show tubercle bacilli or elastic fibers.

Roentgen-ray examination (fig. 9) showed a definite restriction of the movement of the diaphragm on the right. A wedge-shaped area of dulness extended from the right border of the heart to the axilla, obliterating the cardiohepatic angle. The right side of the thorax was less radiant than the left, and the right apex showed some retraction.

As both the physical signs and the roentgen-ray interpretation were suggestive of a thickened pleura or encapsulated fluid at the right base, a thoracentesis was performed, but it was without result. Operative exploration was advised, but was refused by the patient, and she was discharged with the diagnosis resting between a localized empyema and a lung abscess with pleural involvement.

She was seen again in November, 1923, sixteen months later. Her condition had not changed, and further roentgen-ray studies showed the same obliteration of the cardiohepatic angle. She was again discharged untreated.

CASE 5.—F. L., a youth, aged 19, first seen in June, 1922, was referred from the United States Public Health Service with a diagnosis of bronchiectasis. His history included typhoid fever at the age of 10, lobar pneumonia at 15 and again eight months before the onset of his present illness, the removal of adenoids at the

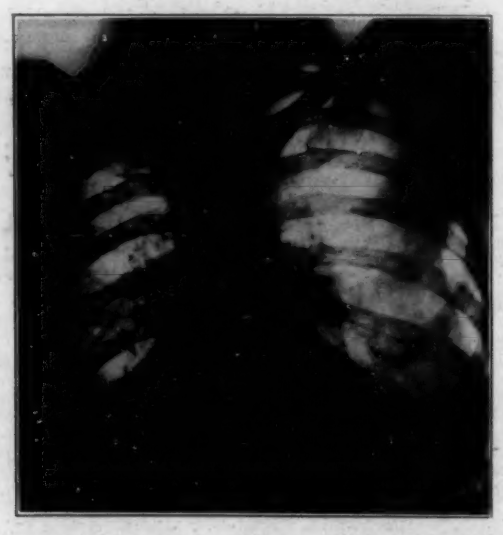

Fig. 9 (case 4).—Atelectasis of the right lower lobe with chronic suppuration.

age of 9 and a tonsillectomy at the age of 16. He had not had any noteworthy cardiorespiratory symptoms before the present illness.

In 1921, the patient was stationed at an army flying field in Texas. His health was excellent until September of that year when, as a result of a "nervous breakdown" from overwork, he was placed in the camp hospital. After three weeks, he developed a "cold" accompanied by a cough and the expectoration of considerable watery, frothy sputum. This cough continued for some weeks, and he was finally discharged from the army in November because of "bronchitis." Returning to his home in Massachusetts, he remained for some time under the care of his family physician, and then entered a government hospital. During this period he raised approximately a cupful of slightly foul sputum a day. Careful studies were conducted to rule out tuberculosis. After several months of observation, during which he showed periods of remission followed by a return of symptoms, he was referred to the Massachusetts General Hospital.

On examination he presented evidence of loss of weight and strength; the breath was foul and there was definite clubbing of the fingers and toes. The right

half of the chest lagged perceptibly during inspiration, and there was impairment of resonance over the entire right lung and, to a less degree, at the left base. At the base of the right lung, at the level of the eighth interspace, a localized area of more than marked dulness was noted. The breath sounds were diminished throughout the right side, but were vesicular except over the area of extreme dulness at the base, where they were intensely bronchial. Numerous musical râles were heard at the bases of both lungs, more marked on the right. The heart did not show enlargement or displacement.

Studies of the sputum failed to show tubercle bacilli or elastic fibers. It was mucopurulent and contained many pneumococci and influenza bacilli.

Fig. 10 (case 5).—Atelectasis of the right lower lobe with chronic suppuration.

A roentgenogram (fig. 10) with fluoroscopic examination showed obliteration of the right cardiophrenic angle and lagging of the right half of the diaphragm. The area of increased density extended downward from the hilum, obliterating the inner portion of the diaphragm, but through it could be traced the outline of the right border of the heart. These observations were interpreted as probably being due to chronic inflammatory changes involving the mediastinal pleura and the inner aspect and lower portion of the lung.

Puncture of the chest failed to reveal the presence of pus, but an exploratory thoracotomy for what was thought to be an encapsulated empyema with a bronchial fistula was performed. In the operation Dr. Wyman Whittemore removed sections of the eighth and ninth ribs and made an extrapleural exploration by stripping the parietal pleura from the wall of the chest. The pleura was not inflamed, and palpation of the lung failed to reveal any thickening or induration.

The patient made an uneventful recovery and was discharged unrelieved, with a diagnosis of bronchiectasis.

CASE 6.—L. D., an Italian girl, aged 9, complained of having had a mild, unproductive cough for six months. During this period she had also had an occasional pain in the chest and night sweats once or twice a week. Three months before admission to the hospital, she was awakened by a slight cough and found her mouth full of bright red blood. When a second hemorrhage of the same type occurred, she was brought to the hospital.

The child was undernourished and showed a slight secondary anemia. She held her head toward the left, apparently due to a spasm of the sternomastoid muscle. The left side of the chest was less prominent than the right but showed good expansion. There was relative dulness throughout the left side of the chest, growing more marked toward the base. The breath sounds on the left became bronchial and more distant from the ear as the base was approached. Just beside the spine at the base the breath sounds were close to the ear. There was marked

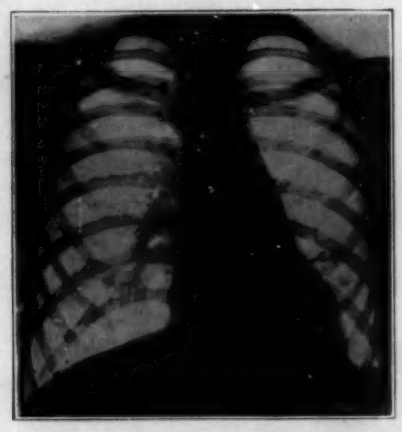

Fig. 11 (case 6).—Atelectasis of the left lower lobe with chronic suppuration. The arrow indicates the border of the lobe which lies behind the heart shadow by which it is nearly obscured. The displacement of the heart toward the affected side aids in the diagnosis.

clubbing of the fingers. Examination of the sputum failed to show tubercle bacilli and the Pirquet reaction was negative.

Roentgen-ray examination showed a high diaphragm on the left, which was limited in its excursion. A triangular area of dulness with a sharply defined border was detected behind the heart. This was interpreted as a localized destructive process near the root and base of the left lung, probably an abscess.

The child was kept under observation for two weeks, during which time her temperature was constantly elevated. A later examination of the chest proved that the small area of egophony and loud tubular breathing beside the spine at the left base disappeared on quiet respiration, only to reappear on coughing or with deep inspiration. This suggested intermittent bronchial obstruction. No notable change took place during her stay in the hospital, and she was discharged untreated with a diagnosis resting between chronic bronchopneumonia and lung abscess.

COMMENT

Bronchoscopic studies have been made in only one of this series of cases, and in the absence of such an examination, the possibility of the presence of the common causes of bronchial obstruction cannot be denied. Chief among these causes are nonopaque foreign bodies, bronchial tumors and strictures, and mediastinal adenopathy. A study of the case records, however, leads us to believe that such conditions can be ruled out in nearly every case. In case 6 it seems that the burden of proof would rest heavily on any one who claimed that a foreign body was not present. The case is included to demonstrate atelectasis of the left lower lobe and also to show the difficulties of its diagnosis. Typical roentgen-ray observations of lobar atelectasis are now considered a definite indication of bronchoscopic examination.

The relation between the atelectasis and the suppurative process is of great interest, but certain aspects must be left undetermined at the present time. A focus of chronic pulmonary suppuration with a discharge of pus into the bronchial tree may produce a secondary atelectasis through bronchial obstruction, as is frequently exemplified by an area of collapse lying peripheral to a deep-seated abscess or pneumonic process.

On the other hand, the possibility that persistent areas of atelectasis may prove to be foci for the development of chronic pulmonary suppuration is suggested by these cases. Four of these six patients date the onset of their symptoms to events which are now known to be at times associated with extensive pulmonary atelectasis; namely, surgical operations (cases 3 and 4), severe trauma (case 2) and postdiphtheritic paralysis (case 1). (These cases were selected on the basis of the roentgen-ray observations in a review of all cases of pulmonary suppuration which came to our attention over a period of three years.)

Primary atelectasis under such circumstances has frequently been described as massive collapse of the lung. Whatever may have been the primary cause, if a lobe such as the ones to which we have referred in the cases of massive collapse were to remain deflated, the mechanism for the origin and maintenance of chronic infection in the retained pool of bronchial secretions is at once evident.

This sequence of events is clearly portrayed by the history in case 4, in which the debilitated patient felt "choked" with mucus after the operation. The sputum was at first thin and watery, but gradually became foul and purulent, presumably as the growth of putrefactive organisms became established. A similar history is frequently reported in cases of the aspiration of foreign bodies when there is known to be stagnation in the distal bronchi and deflation of the corresponding area of lung parenchyma.

This evidence leads us to believe that deflation of a portion of the lung may in itself be a causal factor in the development of a chronic suppurative process. Such deflation has been observed most commonly with the lodgment of a foreign body in a bronchus, but we believe that it may also find origin in the so-called massive collapse of the lung. Atelectasis and fibrosis develop subsequent to the occlusion of a branch of the pulmonary artery, as has been shown experimentally by Schlaepfer.[8] We have observed here one instance in which chronic pulmonary suppuration developed following a postoperative infarction, apparently through these changes rather than through necrosis and sequestration of the involved area. None of the clinical cases cited here gave a history suggesting the occurrence of a postoperative pulmonary infarction of such magnitude.

As the cough and sputum of these patients are apparently due to intrabronchial stagnation in an area of lung which is already deflated, it would seem illogical to employ therapeutic measures which are designed to act by further compression, such as pneumothorax or thoracoplasty. If our concept of the pathologic process is correct, it would seem reasonable to treat these patients by bronchoscopic drainage in an attempt to obtain reexpansion, and if this method fails, by actual resection or destruction of the involved lobe by cautery. It is of interest to note that the patient in case 2 continued to raise over a pint of sputum a day, although the involved area of lung was compressed by the massive hydrothorax which complicated the therapeutic pneumothorax. In case 3 the alleviation of symptoms was greatest following the reexpansion of the lung after the pneumothorax treatment was discontinued, and the final roentgen-ray examination showed reexpansion of the involved lobe.

SUMMARY

The roentgen-ray observations in lobar atelectasis are described.

Six cases of chronic pulmonary suppuration are presented which show lobar atelectasis.

This association may be explained either as atelectasis secondary to a focus of pulmonary suppuration, or, as we suggest, as chronic intrabronchial stagnation superimposed on a primary atelectasis. Such primary lobar atelectasis is found in the so-called massive collapse of the lung, and four of our six patients date their symptoms from incidents known to be commonly associated with this disorder.

8. Schlaepfer, Karl: Ligation of pulmonary artery of one lung with and without resection of phrenic nerve, Arch. Surg. **9**:25 (July) 1924.

Progress in Orthopedic Surgery

THIRTY-SECOND REPORT OF PROGRESS IN ORTHOPEDIC SURGERY *

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CONGENITAL DEFORMITIES

Congenital Dislocation of Hip.—From a study of 349 cases of congenital dislocation of the hip, Engelmann [1] is led to the conclusion that heredity plays a large part in the etiology. He describes the architectural similarity which he found in the pelvic bones of different members of one family, and he believes that the bony features which conduce to mechanically weak joints may likewise be transmitted to descendants. In 253 cases he was able to demonstrate, in addition to the dislocation of the hip, other congenital lesions, most frequently clubfoot.

Ryerson [2] believes that much of the difficulty in maintaining reduction of the hip in infants after manipulation may be avoided by the use of removable celluloid casts. These are not affected by the contamination of urine and feces, may be washed, and possess other obvious advantages over plaster.

Adams [3] describes the obstacles to reduction which were found at operation in a case of bilateral dislocation of the hips in which manipulative reduction had previously been attempted without success. On one side a strong transverse band was found in the front of the capsule,

* This Report of Progress is based on a review of 349 articles selected from 442 titles dealing with orthopedic surgery appearing in medical literature between July 24, 1926, and Nov. 7, 1926. Only those papers that seem to represent progress have been selected for note and comment.

1. Engelmann: Zentralbl. f. Chir. **51**:3242, 1926.
2. Ryerson, E. W.: J. Bone & Joint Surg. **8**:517 (July) 1926.
3. Adams, Z. B.: J. Bone & Joint Surg. **8**:507 (July) 1926.

and this had to be divided before the hip could be reduced. On the other side, bands were found along the inferior portion of the acetabulum, possibly representing a thickened cotyloid ligament. When this band was divided, as well as the reflected tendon of the rectus muscle, the hip was easily reduced. The postreduction roentgenograms showed both femoral heads deep in their sockets.

Galloway [4] reports the results of thirty-eight cases of congenital dislocation of the hip treated by open operation. He feels that the open operation is simple, safe, and reliable, particularly in children under 3 years of age, who have never been subjected to treatment by manipulation or otherwise. He classifies his results as follows: cured, 15; good results, 18; doubtful results, 5; known failures, none. The oldest child in this group was 2½ years old, and the youngest 17 months; none of them had been subjected to previous attempts at manipulative reduction. The author emphasizes particularly that the ideal age for the open operation is from 20 months to 2 years, and that the disturbance of the tissues by previous unsuccessful manipulative efforts renders the complete success of the open operation much more doubtful.

Balensweig [5] reports the end-result in a case of congenital dislocation of the hip in a girl, aged 13. After preliminary traction for three weeks, an open operation was performed. A new acetabulum was made in the upper half of the old shallow acetabulum, the head was reshaped and placed in the new socket. One year later, the patient showed satisfactory function; the shortening was reduced from 2½ inches (6.27 cm.) to 1¼ inches (3.1 cm.); there were 55 degrees of motion in flexion; 10 degrees of abduction and 15 degrees of internal rotation.

[Ed. Note.—The orthopedic world has for some time been looking forward to the publication of end-results in the treatment of congenital dislocations of the hip by Galloway, the apostle of open reduction. The figures which he gives are impressive. There are no failures, and all results are classed as good with the exception of five, which, because they cannot yet be definitely appraised, are classified as doubtful. The group is small when compared with some other groups of end-results that have been published, but we know of none so large without any failures. It is to be noted that the age of the group varied from 17 to 30 months, and the point particularly stressed is absence of previous attempts at reduction. The results are less good when operation is performed only after manipulative failure. The absence of mortality attests the skill of the operator. It seems to us that Galloway's results support his contentions and that they constitute a challenge to the advocates of manipulative reduction.]

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4. Galloway, H. P. H.: J. Bone & Joint Surg. **8**:539 (July) 1926.
5. Balensweig, I.: J. Bone & Joint Surg. **8**:534 (July) 1926.

Congenital Absence of the Tibia.—Congenital absence of the tibia is one of the rarest congenital malformations and the rarest malformation of the lower limb. According to Evans and Smith,[6] the number of recorded cases is now 105. Absence of the tibia is rather more commonly total than partial, unilateral than bilateral, and right-sided than left-sided. Other abnormalities are frequently associated, but rarely is there any evidence of fetal disease. Disarticulation at the knee, followed by the application of a peg leg or artificial limb, the authors believe, is the best line of treatment. Conservative operations are indicated only in cases of partial defect, when the gap is small and limited to the middle portion or distal end of the tibia.

[ED. NOTE.—Although disarticulation at the knee usually gives a less satisfactory functional result than amputation above the femoral condyles, in this type of case it would appear to be the operation of choice on account of the necessity of saving the lower femoral epiphysis for the sake of growth. If necessary, reamputation can be performed after the patient reaches adult life.]

TUBERCULOSIS

Paraplegia with Pott's Disease.—Sorrel and Sorrel-Dejerine[7] have made a study of forty-two cases of paraplegia caused by Pott's disease in children and adults. On the basis of the clinical picture and the evolution of the disease, they were able to distinguish three different types of paraplegia. The first is the transient type, which clears up in a few weeks or months. It is of rare occurrence. The second type is also curable, but may persist for from eighteen to twenty-four months. It appears at an early stage of the tuberculous spondylitis, the development of the paraplegia is rapid and complete, and the clinical picture appears grave. Nevertheless, with proper treatment the paralysis disappears without any sequelae. The third form is an incurable paraplegia and appears as a late complication, often three years after the onset of the disease. It develops slowly and is never complete. It is compatible with a long existence. The curable forms are observed in adults as well as in children. They are induced by intraspinal abscesses, while the incurable form is the result of an external pachymeningitis, which causes so much destruction in the spinal cord that restoration ad integrum is impossible. The transient type of paraplegia is probably caused by edema of the dura mater without the formation of an abscess. The authors consider the prognosis more grave in a paraplegia starting with a mild clinical picture than in one with severe and rapidly developing manifestations.

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6. Evans, E. L., and Smith, N. R.: Arch. Dis. Childhood **1**:194 (Aug.) 1926.
7. Sorrel, E., and Sorrel-Dejerine: Presse méd. **34**:785 (June 23) 1926.

A pathologic study of four specimens of tuberculous disease of the spine with abscess, recovered at autopsy, has convinced Willis [8] that the lesions of the vertebrae are more extensive than has been claimed. In addition to the destruction of the bodies of the vertebrae, there is frequently involvement of the articular and transverse processes. There may be diffuse extension of the disease without any kyphosis or other deformity. Coexistent with extensive destruction of bone, there may be an appearance in the roentgenogram of merely superficial erosion. Productive changes in the bone are to be found in active lesions as well as in old healed spines. He urges the necessity for a thorough roentgen-ray examination and a search for multiple lesions.

End-Results of Tuberculosis of the Knee Joint.—Martens [9] has made a study of 211 patients with tuberculosis of the knee joint who were treated during a period of ten years at the surgical clinic in Goettingen (Stich). Eighty-nine patients were treated without operation, 105 patients had resection of the knee, and in eleven cases primary amputation was performed. The nonoperative treatment consisted of fixation by plaster casts, and, in certain cases, of aspiration and the injection of iodoform. Of the patients so treated, eighteen were cured with restoration of good function, six were cured without ankylosis, and twenty-one showed poor results. Of the cases of resection, eighty-eight patients were cured, although some required second operations; twenty-four patients were not cured, underwent secondary amputations, or died. The late results were good in sixty-four cases and poor in eleven cases. Only six patients showed shortening of more than 5 cm. after the resection. The percentage of cures is 77 per cent. He concludes that the decision as to whether a patient should be treated operatively or nonoperatively is an individual problem which must be settled on its merits in each case.

[Ed. Note.—We note that a much higher percentage of end-results was obtained in the cases in which there were operations than in the cases in which operations were not performed. One cannot, therefore, make an accurate comparison between the two groups. It is also to be suspected that the diagnosis may have been in error in a certain number of the eighteen cases in which the patients are reported cured with good function.]

New Method of Hip Function for the Treatment of Tuberculosis of the Hip Joint.—Hibbs [10] describes a new method for extra-articular fusion of the hip joint. The essential feature of this method is a transposition of the trochanter so that it makes contact with the ilium above

8. Willis, T. A.: Surg. Gynec. Obst. **43**:285 (Sept.) 1926.
9. Martens, Fr.: Beitr. z. klin. Chir. **135**:631, 1926.
10. Hibbs, R. A.: J. Bone & Joint Surg. **8**:522 (July) 1926.

and with the femur below, securing continuity of periosteum. He reports the results in the first twenty cases; eighteen of these patients have definite fusion, shown by physical and roentgen-ray examination. The ages of the patients vary from 4 to 31 years; the first was operated on April 5, 1923. The average duration of the disease before operation was eight and one-half years; the shortest, six months; the longest, twenty-six years. The one death was that of a man, aged 21; he died nine months after operation from miliary tuberculosis. These twenty patients had been treated at the New York Orthopedic Hospital for an average of six years, and some had been treated for years in other hospitals. Six had been treated at the country branch for periods of from three to seven years. In cases of severe flexion and adduction deformity, this is not corrected at the time of the operation. Subtrochanteric osteotomy was performed in one case and may be necessary in others, but not until the extra-articular fusion is complete.

POLIOMYELITIS

A Milk-Borne Epidemic.—Knapp, Godfrey and Aycock[11] have made a study of an epidemic of poliomyelitis which occurred in the city of Cortland, N. Y., the circumstances of which pointed strongly to a contamination of milk supply as the cause. From Dec. 14, 1925, to Dec. 25, 1925, eight cases developed. These were the first to occur subsequent to the appearance of three cases more than two months before. All the patients were found to have consumed milk from the same dealer. On December 7, seven days before the onset of symptoms in the first patient of this group, a 16-year old boy who was working on the dairy farm milking the cows and handling the milk, became ill with poliomyelitis. The time which elapsed between the first mild appearance of his symptoms and the development of the later cases is consistent with the generally accepted minimum and maximum incubation periods of poliomyelitis. While this outbreak points strongly to transmission through the milk, the authors are not of the opinion that this is the usual mode of spread of the disease.

The Kansas City Epidemic of 1925.—Diveley,[12] in reporting the epidemic of infantile paralysis of 1925 in Kansas City, states that fifty-one cases had been reported after the lapse of four months. Of these, seventeen, or 33 per cent, died; two, or 4 per cent, recovered without residual paralysis, and nineteen, or 37 per cent, showed paralysis of varying degrees, and all the latter group have residual paralysis at the present time, about 50 per cent reporting improvement. Diveley feels that the patients treated with Rosenow's serum showed a more rapid

11. Knapp, A. C.; Godfrey, E. S., and Aycock, W. L.: Outbreak of Poliomyelitis, Apparently Milk Borne, J. A. M. A. **87**:635 (Aug. 28) 1926.

12. Diveley, R. L.: J. Missouri M. A. **23**:237 (July) 1926.

recovery, and that the paralysis was not so profound or so extensive as in the untreated patients. The effect of spinal drainage on the acute symptoms was almost phenomenal, the symptoms disappearing for the most part a short time after the drainage and appearing only when the spinal pressure again rose above normal. The death rate in the series of patients who were treated was much smaller than that in the group of untreated patients. There was a glycosuria and hyperglycemia in a large percentage of the cases, thus substantiating the point made by several observers that this infection has some effect on the carbohydrate metabolism, the mechanism probably being an inflammation of the pituitary bodies. Diveley advises as the best treatment strict isolation, early and repeated spinal drainage to keep down abnormal pressure, and serum, either human or the immunized horse serum of Rosenow, given immediately after the spinal drainage, either intravenously or intramuscularly, depending on the severity of the symptoms.

SYPHILIS OF THE BONE

Dillingham and McCafferty[13] describe three cases demonstrating various phases of syphilis of the bone. One patient showed an early syphilitic periostitis. The second presented a syphilitic osteomyelitis of the cortical bone and a thickening of the periosteum with destruction of the soft parts overlying the clavicle. The third was one of extensive osteomyelitis, with thickening of the cortex and a definite periostitis with evidence of sinus formation and sequestrum.

PYOGENIC INFECTIONS

Acute Hematogenous Osteomyelitis.—Krasnobaew,[14] in discussing the subject of acute osteomyelitis, said that to him the importance of the problem arose from the great number of patients whom he had had to treat in his service during the twenty-one years from 1904 to 1925. The total number of cases was about 600, of which 428 were acute. From a careful study of the cases and of his results, he arrives at the following conclusions:

1. It is of special importance to consider the general condition of the patient, to take account of his vitality, and to have the greatest care for the tissues involved. 2. In the severe cases of acute osteomyelitis it is necessary to avoid, in the first stage of its evolution, all operations requiring general anesthesia. 3. Too radical treatment in the first stage of acute osteomyelitis is not only useless, but dangerous. 4. In the first septic period of osteomyelitis it is necessary to refrain from doing more than opening the abscess as early as possible by incisions reaching to the

13. Dillingham, F. H., and McCafferty, L. K.: Am. J. Syph. **10**:373 (July) 1926.

14. Krasnobaew, T.: Chir. d. org. di movimento **10**:537, 1926.

bone. 5. Subsequently, the treatment of the inflammation requires all attention. 6. Gauze packs and tampons should not be used. Drainage tubes should be rarely used and only for a short time. 7. In cases of osteomyelitis of the diaphysis, involvement of the joint frequently develops from lack of proper care of the wound. 8. If the wounds are well treated, amputation or resection is rarely necessary. 9. At the end of the septic period, without waiting for the formation of an involucrum, sequestrectomy should be performed and all the necrotic parts removed. The wound should be sutured. 10. Motion is especially dangerous when osteomyelitis of an epiphysis is accompanied by suppurative arthritis. 11. In cases of epiphyseal osteomyelitis of infants, especially when the suppuration has not yet invaded the joint, one should use repeated aspirations by means of a syringe. 12. In the severe cases in infants, when the aspirations are insufficient; in adolescents, and also in cases in which the pus has invaded the capsule, incisions and arthrotomies are necessary. The incisions need not be large. In these cases, also, gauze packs and tampons are to be avoided. 13. The necessity of resection occurs only in cases of epiphyseal osteomyelitis of the hip, and even here it is necessary to resect only in rare cases in which there is necrosis of the epiphysis with separation of the epiphysis from the diaphysis.

Acute Suppurative Arthritis.—Caldwell,[15] in discussing the treatment of well established suppurative arthritis, gives the following advice. Aspiration, with or without the injection of antiseptics, is inadequate. Arthrotomy by one or more incisions, adequately large, is indicated as soon as diagnosis is made. Intracapsular drains are dangerous and superfluous. Immediate, active mobilization following arthrotomy appears to be unnecessary, to say the least of it. Since the results of the so-called Willems method are certainly no better than, if as good as, those in which arthrotomy has been followed by fixation, it would seem wiser to abandon it for the simpler, more logical, more truly orthopedic and surgical procedure of arthrotomy followed by fixation.

[ED. NOTE.—On the basis of previously published reports and our own experience, we feel that there is a place for repeated aspiration as a method of treatment in certain mild cases of suppurative arthritis. Nor do we agree with Caldwell that complete fixation is desirable as a routine measure. We believe that in certain cases mobilization should be encouraged as early and as actively as possible.]

DISEASES DUE TO NUTRITIONAL DISTURBANCES

Rickets.—Cozzolino[16] reports a case of congenital myxedema and discusses the possibility of this disease coexisting with rickets. He states that most writers admit that the diseases which interfere with or inhibit

15. Caldwell, G. A.: South. M. J. **19**:637 (Aug.) 1926.
16. Cozzolino, O.: Pediatria **34**:761, 1925.

the regular growth of the nursing child prevent the development of rickets. There are a few authors who refuse to accept this. Cozzolino does not believe that any proof of the impossibility of association of myxedema and rickets has yet been brought forward, but care must be exercised in making such a diagnosis if the sign on which the diagnosis of rickets is based is craniotabes. An error may creep in here, as it has not been absolutely proved that craniotabes is pathognomonic of rickets.

Experiments made by Maslow, Shelling and Kramer [17] demonstrate that antirachitic properties can be imparted to orange juice by irradiation with the quartz mercury vapor lamp. Healing may be demonstrated as early as five days after feeding irradiated orange juice to rachitic rats, and is almost complete at the end of fifteen days.

Comparison of Antirachitic Potency of Irradiated Cod Liver Oils.—A medicinal cod liver oil of known origin, known chemical and physical characteristics, and of vitamin A potency, was irradiated by Wyman and his group of co-workers [18] under carefully controlled conditions for periods varying from one-half to two hours. The antirachitic potency of the original oil and of the irradiated oils was studied to determine to what extent the antirachitic activity of the original oil was enhanced by irradiation with the ultraviolet ray. The results obtained indicate that there was little if any difference in the antirachitic potency of the original oil and that which had been irradiated.

Osteitis Fibrosa Generalisata.—Mandl,[19] after discussing the possibility of relationship between the parathyroid bodies and generalized osteitis fibrosa, reports the case of a man, aged 39, who was suffering from an advanced, severe form of that disease. Examination of the urine showed a marked increase above normal in the quantity of calcium that was being excreted. Excision of a small tumor of one of the epithelial bodies resulted in marked improvement. The excessive elimination of calcium salts in the urine was stopped. There was improvement, both subjective and objective, with increase in weight, and the roentgen-ray examination showed a marked increase in the lime content of the bone.

Hypertrophic Osteo-Arthropathy.—Ramond and Bascourret [20] discuss a case of hypertrophic osteo-arthropathy of Pierre Marie. The patient, a policeman, aged 26, complained of an abnormal and progressive

17. Maslow, H. L.; Shelling, D. H., and Kramer, P.: Bull. Johns Hopkins Hosp. July, 1926, p. 56.

18. Wyman, E. T.; Holmes, A. D.; Smith, L. W.; Stockberger, D. C., and Pigott, M. G.: Boston M. & S. J. **195**:525, 1926.

19. Mandl, F.: Zentralbl. f. Chir. **53**:260, 1926.

20. Ramond, L., and Bascourret: Bull. et mém. Soc. méd. d. hôp. de Paris **50**:1015, 1926.

enlargement of the hands and feet, with slight pain and crepitation in the knees, ankles and wrists, and a sense of general fatigue. The condition had had its onset four years previously. The examination showed osteo-articular deformities localized at the extremities, characteristic appearance of the fingers with incurvation of the nails. The picture on roentgen-ray examination was typical. In discussing the origin, which is chiefly from pulmonary diseases, Léri states that small chronic lesions of sclerosing tuberculosis are able to give the syndrome.

Dwarfism with Pluriglandular Syndrome.—Bénard, Hillemond and Laporte [21] present a case of dwarfism with pluriglandular syndrome. The striking feature was the small stature with remarkable shortness of the extremities, which led them to suggest the term "nanisme acromicrique" in contrast to "gigantisme acromégalique." The patient was a girl, aged 17, with a complexity of symptoms of myxedema, with pigmentation and sclerodactylia. The condition was different from the micromelia of Galliaud and Levy, the dyschondroplasia of Ollier and the pituitary dwarfism described by Erdheim.

DISTURBANCES OF GROWTH

Osteochondritis of the Epiphyses.—Christie,[22] discussing the whole subject of epiphyseal osteochondritis, states that there is little doubt that the etiologic factors and pathologic changes are similar in Legg's disease, Schlatter's disease, Koehler's disease and vertebral epiphysitis. The same disease also occurs in the epiphyses of the os calcis and of the olecranon. There is also fairly good evidence that the osteochondritis dissecans of König and the rare condition known as malacia of the carpal semilunar bone belong in the same category. Regarding the etiology of epiphyseal osteochondritis, Christie states that there is no agreement, except that it is definitely known that the disease is not tuberculous, rachitic or syphilitic. There is a little evidence to support an infectious theory, but the cases in which operation has been performed and in which a possible causative organism has been isolated, are so few that they may be explained on the grounds of coincidence or of accidental contamination. The theory of an endocrine origin is a pure assumption with no reliable evidence to support it. A history of trauma is present in some of the cases, but not in a sufficient proportion to make it appear to be the ultimate cause. The supposition has been advanced that the condition is primarily due to some developmental anomaly or constitutional disturbance which eventually results in poor nourishment of the epiphysis at the time of its more rapid development. It is worthy of note that each lesion is associated with a definite age period, and that the period is the

21. Bénard, R.: Bull. et mém. Soc. méd. d. hôp. de Paris **50**:967, 1926.

22. Christie, A. C.: Osteochondritis or Epiphysitis; Review, J. A. M. A. **87**:291 (July 31) 1926.

one in which the affected nucleus of the bone is undergoing its most active development. The opinion of the majority of investigators inclines to the idea that the disease is due to long continued or occasionally acute trauma, resulting in interference with the circulation and in subsequent necrosis involving both the bone and the cartilage of the epiphysis.

Coxa Plana.—Valle and Verbrugge [23] report some observations on the rôle of heredity in coxa plana and kindred conditions of the hip. Their studies lead them to conclude that both heredity and age are important factors. One may inherit a predisposition to these conditions, accompanied or not by actual malformation. There is a potentiality of disease which under the influence of trauma, of infections, of nutritive disturbances or of endocrine disorders may produce in the period of childhood osteochondritis or coxa vara; in youth, coxa vara of adolescence, and in adult life, arthritis deformans.

Case of Legg-Perthes' Disease.—Kidner [24] reports a case of what he regards as Legg-Perthes' disease. The history began with weakness of the left hip, observed by a mother in an infant. The child walked late, and gradually developed a limp; there was only slight pain. When first seen, at the age of 8 years, there was 1 inch (2.5 cm.) shortening, and the motions were limited to 15 degrees in all directions. Roentgen-ray examination showed spina bifida occulta of the fifth lumbar, deformity of the head and neck of the femur and of the acetabulum, characteristic of Legg-Perthes' disease. Neurologic examination showed complete anesthesia of the anterior two thirds of the thigh and lower leg, hyperesthesia of the posterior third. A biopsy was performed, and the cartilage appeared smooth and apparently normal. A section of the head was examined by Dr. W. B. Phemister, who found both degenerative and reparative changes, but no evidence of inflammation. He regarded the process as a circulatory disturbance and considered it a case of Legg-Perthes' disease, despite the early age of onset.

[ED. NOTE.—We would have some hesitation in calling this Legg-Perthes' disease in view of the history and of the neurologic disturbance, nor do we believe that the pathologic picture of Legg-Perthes' disease has yet been sufficiently established to permit the observations on study of the specimen to be adduced as proof. Is it not possible that a neurotrophic process affecting the hip might give the same appearance?]

Apophysitis of the Os Calcis.—O'Ferrall [25] believes that both trauma and infection play a part in the etiology of apophysitis of the os calcis. Many cases are undoubtedly not recognized, and recover under the treat-

23. Valle and Verbrugge: Chir. d. org. di movimento **10**:364, 1926.
24. Kidner, F. C.: J. Bone & Joint Surg. **8**:565 (July) 1926.
25. O'Ferrall, J. P.: South. M. J. **19**:549 (July) 1926.

ment of simple sprain. His conclusions are as follows: Apophysitis of the os calcis is most common in growing boys during the period of their greatest growth activity. The condition clinically is usually unilateral, but by roentgen-ray examination the epiphysis on both sides is often found to be involved. Close questioning may reveal that bilateral symptoms have existed, one side being more affected than the other. Freedom from weight-bearing, fixation of the extremity in a plaster cast to lessen the pull of the Achilles tendon and the administration of cod liver oil usually relieve the condition promptly and permanently.

[ED. NOTE.—We are not at all convinced that it is necessary to employ plaster fixation in the treatment of this condition. The symptoms are usually mild and are promptly relieved by adhesive strapping and elevation of the heel. We have seen no bad results, even in those patients who have been totally untreated.]

Symmetrical Disease of the Epiphyses of the Hand.—Kloiber [26] describes a symmetrical disease of the middle joints of the third fingers of both hands which is accompanied by a slight disturbance of the function. Clinically, there is slight thickening, and the roentgen-ray examination shows a marked change in the epiphysis of the second phalanx. The epiphysis is diminished in length, but markedly increased in width. The epiphyseal line is irregular and thicker than normal. The articular gap is narrowed and in some places seems to have disappeared. The nucleus is split in two or three fragments. The disease closely resembles Koehler's disease of the metatarsal head, particularly in regard to the destruction of the epiphysis, the enalargement of the metaphysis and the slight disturbance of function.

Significance of Accessory Tarsal Scaphoid.—Zadek [27] discusses the significance of the accessory tarsal scaphoid in its relation to weak feet. He has operated on three such cases and found an abnormal insertion of the tibialis posticus, the tendon being entirely or mainly inserted into the scaphoid. This results in muscular imbalance and leads to weak feet. Such cases do not respond to supportive or exercise treatment. Good functional results are obtained by removal of the accessory scaphoid and of the prominent portion of the scaphoid if accompanied by astragalo-scaphoid arthrodesis.

ARTHRITIS

Classification and Treatment of Chronic Arthritis.—Cecil and Archer,[28] after four years' experience with chronic arthritis in the Cor-

26. Kloiber: Fortschr. a. d. Geb. d. Roentgenstrahlen **34**, no. 4, 1926.

27. Zadek, I.: J. Bone & Joint Surg. **8**:618 (July) 1926.

28. Cecil, Russell L., and Archer, Benjamin H.: Classification and Treatment of Chronic Arthritis, J. A. M. A. **87**:741 (Sept. 4) 1926.

nell Clinic, are convinced that this disease occurs in two distinct and fundamentally different clinical forms. They prefer the classification of Nichols and Richardson, feeling that the division into the two groups, proliferative and degenerative, rests on definite pathologic and probably etiologic grounds. They feel that the proliferative form is almost certainly infectious in origin and that the degenerative type is noninfectious. They are not able to exclude the possibility of some toxic factor playing a part in the causation of degenerative arthritis. Largely on the basis of clinical manifestations, they differentiate three subdivisions of each group:

I. Proliferative Type Arthritis: (*a*) chronic infectious arthritis; (*b*) specific arthritis; (*c*) true arthritis deformans, a chronic progressive polyarthritis of unknown origin. II. Degenerative Type Arthritis: (*a*) arthritis of the menopause; (*b*) degenerative monarticular arthritis (morbus coxae senilis); (*c*) senile arthritis. In the proliferative type different infectious agents are probably responsible for the different clinical manifestations. The three subdivisions of degenerative arthritis are largely a matter of clinical convenience, though here, again, a variety of etiologic factors may play a part. It seems probable, for example, that arthritis of the menopause is dependent on some endocrine disturbance. In a study of 612 cases of chronic arthritis, approximately two thirds (68 per cent) were of the proliferative type and one third (30 per cent) were of the degenerative type. Proliferative arthritis occurs most frequently in young persons; degenerative arthritis is more often seen in the middle-aged and elderly. The commonest form of proliferative arthritis is associated with focal infection about the teeth or tonsils. The commonest variety of degenerative arthritis is the arthritis of the menopause. The proliferative arthritis is presumably an infectious process. Degenerative arthritis does not appear to be of infectious origin. It appears more likely that it is, as its name implies, a degenerative process analogous to arteriosclerosis and to the other degenerative changes that attack various organs in old age. The essential feature in the treatment of proliferative arthritis is the removal of all foci of infection. In degenerative arthritis the treatment should be directed chiefly toward accelerating metabolism by means of the iodides, physiotherapy and other appropriate measures. Many of these patients are improved by a low caloric diet.

[Ed. Note.—We think it is of interest to note that the conclusions of Cecil and Archer are in line with those of nearly all observers who have had the opportunity of making an intensive study of chronic arthritis. There are two distinctive main types of arthritis in the opinion of most authorities, and the subdivisions made by Cecil and Archer may be recognized under other names used by different writers.]

Diet in Treatment of Chronic Arthritis.—Pemberton [29] says that it is common knowledge that even in health a great intake of food may induce hebetude and lethargy probably through an influence on the flow of the blood, and there is reason to believe that it is through an influence on the flow of the blood that a curtailment of diet exerts part of its effect in arthritis. In view of the undoubted benefit of measures which improve the flow of the blood and hasten metabolism, such as exercise, massage and external heat, it seems likely that at least part of the influence of a reduced diet results from placing a lessened demand on the machinery of exogenous metabolism, especially in the muscles. Pemberton says that the chief considerations in dietetics are: first, to reduce the total caloric intake to suit the individual case—in general, to about 30 Kg. of body weight according to the usually accepted approximate requirements for rest; and second, in diets of relatively high caloric value, to redistribute the foodstuffs so that the proportion of calories is larger from fats than in the usual dietary and less from carbohydrates. Pemberton feels that diet constitutes the best method of meeting the therapeutic indications in chronic arthritis.

Vaccine Therapy and Serologic Diagnosis in Arthritis.—Burbank [30] reviews the history of vaccine therapy in arthritis and outlines his own method of procedure. The isolation and identification of the organisms is the most difficult task, and for this process careful serologic methods of study are all-important. Vaccine treatment, when properly prepared, controlled and administered, represents, in his opinion, the most potent method of attacking this disease. To treat a patient adequately it is also important to see that the hygiene and environment are rendered as favorable as possible. He believes that favorable results may be obtained in practically all patients whose serologic reactions show favorable complementary values, if they have not undrained infectious foci, are not suffering from any debilitating disease, and have the patience to continue the treatment over considerable periods.

Rôle of Dysentery Bacillus in Arthritis Deformans.—Clifford [31] reports his studies of seven cases of arthritis deformans of type I, in four of which there was evidence either of an active or of a partial infection with *Bacillus dysenteriae* of Flexner. He feels that dysentery should be considered as an etiologic factor in this form of arthritis and ruled out only after bacteriologic studies and agglutination reactions, using a complete series of antigens. He does not feel that his observations cover a series of cases large enough to permit any more definite conclusions.

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29. Pemberton, R.: New York State J. M. **26**:668 (Aug. 1) 1926.
30. Burbank, R.: J. Bone & Joint Surg. **8**:657 (July) 1926.
31. Clifford, S. H.: Rôle of Dysentery Bacillus in Arthritis Deformans, Am. J. Dis. Child. **32**:72 (July) 1926.

Rheumatic Nodules.—Coates and Coombs [32] have made a study of rheumatic nodules. They describe the tiny granules which are found in the subcutaneous tissue of the rheumatic child and discuss their significance. The constitution of rheumatic nodules obtained from patients with rheumatoid arthritis, Still's disease and endocarditis lenta is described. They are shown to be histologically identical with those found in orthodox rheumatic infection.

TUMORS OF BONE

Lesions of Bone.—Bloodgood [33] gives a brief review of different types of lesions of bone based on a study of about 1,000 cases. He states that there is a small but increasing number of cases of sarcoma of the bone in which the patients have remained well for five years or more after amputation, cases which have been accepted under Codman's valuable registry of bone tumors. The fact that the diagnosis is being made at an earlier period is the one explanation of these better results. The improvement in diagnostic ability is undoubtedly due to the interest stimulated by the registry. Sarcoma is greatly on the increase. Bloodgood feels that many patients formerly died at home without record. Now practically all reach some clinic, where a roentgen-ray examination is made. He says that the greatest increase has occurred in certain types of lesions of the bone: healed bone cysts, Paget's disease, sclerosing nonsuppurating osteomyelitis, and a form of destructive osteomyelitis, often multiple, but when occurring as a single lesion difficult to differentiate from sarcoma and multiple exostoses. Syphilitic lesions of bone are decreasing. The number of amputations for giant cell tumor has decreased far beyond expectations. A limb is seldom amputated unnecessarily. Radiation is being given a fair trial, and the dangerous biopsy is becoming less frequent.

Operability of Bone Tumors.—Meyerding [34] says that the operability of a bone tumor depends on its local and general characteristics. Whether malignant or benign, the type, size or situation may prohibit surgical removal. In certain benign cases if the growth is accessible, operation may offer hope of a permanent cure. In doubtful cases, operation affords the means of definite diagnosis, and in case of malignancy removal offers palliative relief, if not more. The age and sex of the patient, size and origin of the tumor, its penetration through or invasion of periosseous tissue, its proximity to important structures, the danger of fracture and the general physical condition of the patient are all factors which must be taken into account in determining the operability.

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32. Coates, V., and Coombs, C. F.: Arch. Dis. Child. **1**:183 (Aug.) 1926.
33. Bloodgood, J. C.: South. M. J. **19**:541 (July) 1926.
34. Meyerding, H. W.: Radiology **7**:29 (July) 1926.

Pulsating Tumors in Bone.—Alessandri [35] reports his observations on three cases of pulsating tumors of bone and one nonpulsating tumor caused by metastasis from hypernephroma. He has collected from the literature additional cases to make ninety-two in all, of which eighty-six were hypernephroma and six were tumors of the suprarenal capsule. His conclusions are as follows: 1. Metastases in the skeleton may come from neoplasms of various organs, but the most common are from epithelial tumors of the breast, prostate, thyroid and suprarenal gland. 2. Pulsation sometimes occurs in metastatic tumors from the thyroid, but more frequently in those from the suprarenal gland. Eighteen of these have been observed. 3. In thyroid tumors the metastases may be exclusively in the skeleton and may be single. The metastases from suprarenal tumors may also be single. 4. In suprarenal tumors the metastases may appear early, even before the most careful examination reveals any tumor in the region of the kidney.

STRAIN OR PAINS IN THE BACK

Rôle of the Lumbar Ligaments in Back Strain.—Lowman,[36] writing of the iliolumbar ligaments of the spine, points out that these structures. aside from exerting a lateral, stabilizing effect, are also suspensory in function and act as a second line of defense in protecting the back from undue strain. If there is any failure of the sacro-iliac ligaments to support that joint, there must occur increased tension on all the other structures involved in supporting or stabilizing the spinal column. In the tilting forward of the pelvis on one side or the other, with a sudden twisting strain, the stress thrown on one of the iliolumbar ligaments is sufficient to produce a sprain, the symptoms being severe pain in the back and lateral deviation of the spine. In patients with hollow back and with the sacrum tilted forward, it can be readily seen that the sacrum bears less weight and these ligaments more because of their direct suspensory function. Evidence of the strain which these ligaments bear is to be found on roentgen-ray examination in the spur formation and calcification of these tissues.

Traumatic Spondylolisthesis.—Henry [37] reports three cases of traumatic spondylolisthesis, in which the patients dated their symptoms back to falls, several years previous, in which they landed in the sitting position. They returned to work within a few weeks after their accidents, but complained of pain originating at the time of injury. Henry states that the four cardinal signs of spondylolithesis are: (1) a prominent, vertically placed sacrum; (2) a palpable, visible hollow above the sac-

35. Alessandri, R.: Policlinico **33**:273, 1926.

36. Lowman, C. L.: Rôle of Iliolumbar Ligaments in Low Back Strains, J. A. M. A. **87**:1002 (Sept. 25) 1926.

37. Henry, M. O.: Minnesota Med. **9**:376 (July) 1926.

rum; (3) a sharp lordosis of the lower lumbar spine, and (4) limitation of extension in the lumbar spine. In all three of Henry's cases a fusion operation was performed on the spine. All the patients made a good recovery, and get along well at present, although they must avoid heavy lifting, bending and stooping.

Bone Graft for Sacro-Iliac Fixation.—Verrall [38] describes a new method for fixation of the sacro-iliac joint by means of a tibial bone graft. The graft is driven through drill holes in the posterior iliac spines and crosses transversely at the back of the sacrum, its middle portion coming in contact with the base of the second sacral spinous process and lying under the erector spinae muscles. The author has performed the operation in four cases, with satisfactory results. The first two patients were treated on plaster beds for five and three months, respectively. In the third case a spinal frame was used for the after-treatment, and in the last case no splint was used at all.

[Ed. Note.—The operation appears less sound from the mechanical and physiologic standpoint than other operations now in use.]

MISCELLANEOUS

Osteomyelitis Variolosa.—The occurrence of lesions of the bone in smallpox has been pointed out from time to time by various writers, but such lesions have been observed heretofore only in the late stages. It has remained for Huenekens and Rigler [39] to record the changes in the acute period. They report the case of a child ill with smallpox who also presented a picture of acute polyarticular arthritis. The joint symptoms in the upper extremities developed shortly after the appearance of the eruption, and the involvement of the other joints followed rapidly. Roentgen-ray examination revealed widespread destructive processes located at the epiphyseal lines of the long bones and in the first cervical vertebra, which dislocated spontaneously. The acute condition in the bones continued for two months after the eruption had disappeared and was accompanied by fever and leukocytosis. Repair finally occurred with almost complete recovery except for the unreduced dislocation of the neck. The rapidity of the onset, the symmetrical, widespread dissemination and involvement of a vertebra, together with absence of suppuration, all coincide with Chiari's observations. The selective nature of the process, the lesions being confined to the region of growth in the bone, is in accordance with the ideas of Musgrave and Sison, based on their observation of the late deformities.

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38. Verrall, P. J.: J. Bone & Joint Surg. **8**:491 (July) 1926.

39. Huenekens, E. J., and Rigler, L. G.: Osteomyelitis Variolosa, Acute Stage, J. A. M. A. **87**:295 (July 31) 1926.

Melitococcus Spondylitis.—Roger [40] of Marseilles calls attention to the frequent occurrence of spondylitis in Malta fever. He reports vertebral lesions of a characteristic type in about 30 per cent of the cases.

Chronic Osteitis of Semilunar Bone (Kienböck's Disease.).—Henderson [41] reports two cases of osteitis of the semilunar bone, or Kienböck's disease. The etiology of this condition is generally thought to be traumatic, either a definite injury or repeated trauma incidental to occupation. Kienböck's syndrome is characterized by three stages: (1) the acute, lasting possibly only a few hours, coming on immediately after the injury, rarely lasting more than a few days, and generally not more than a few weeks; (2) the period of freedom from pain and disability, sometimes lasting as long as two months; (3) the period of actual disease, in which the osteitis definitely assumes form and persists with symptoms, perhaps for years. Kümmell's disease is the nearest analogy. Roentgenograms immediately after the injury do not show abnormalities, but later show varying degrees of osteitis. The author advised against operation in his cases, treating the patients by splinting.

Spontaneous Dislocation of the Biceps Tendon.—Meyer,[42] an anatomist, has observed in the dissecting room four cases of complete dislocation of the tendon of the long head of the biceps brachii, three in men, one in a woman, out of 286 cadavers, or 1.25 per cent. The tendon was not adherent and was wholly preserved, even when the entire superior portion of the articular capsule was completely destroyed. All the subjects were elderly, and there were well marked senile changes in the bone and joint capsule. In regard to the genesis of this dislocation, Meyer thinks it can be produced only after destruction of the superior portion of the articular capsule and then only in the position of marked abduction and lateral rotation, facilitated perhaps by flexion of the forearm in the supinated position. Such conditions would produce a maximum of relaxation of the tendon and permit it to be carried forward over the lesser tuberosity. Once dislocated it would probably always remain, especially if continued attrition in the region of the tuberosities led to bony proliferation and obliteration of the intertubercular sulcus.

Spontaneous Dislocation of the Hip.—Jones [43] has studied a series of twenty-seven spontaneous or pathologic dislocations of the hip. These are classified as follows: (1) dislocation from muscular paralysis; anterior poliomyelitis, six cases; spastic paraplegia, one case; (2) dislocation from acute arthritis; septic arthritis, three cases; pneumococcal arthritis, three cases; arthritis of exanthemas, two cases; pyemic arthritis, one case;

40. Roger, H.: Presse méd. **34**:929 (July 24) 1926.

41. Henderson, M. S.: J. Bone & Joint Surg. **8**:504 (July) 1926.

42. Meyer, A. W.: Spontaneous Dislocation of Tendon of Long Head of Biceps Brachii, Arch. Surg. **13**:109 (July) 1926.

43. Jones, R. W.: Brit. J. Surg. **14**:36 (July) 1926.

arthritis of doubtful origin, three cases; acute tuberculous arthritis, two cases; (3) dislocation from chronic arthritis, none; (4) subluxation from chronic arthritis: tuberculous arthritis, two cases; osteo-arthritis, three cases; hemophilia, one case. The author believes that the underlying pathology is essentially the same in all cases, a muscular derangement resulting in extreme flexion and adduction. This is unquestionably true in the paralytic cases; in the arthritic cases the inflammatory process alone does not bring about dislocation unless accompanied by abductor and flexor spasm. The deformity must be one of both flexion and adduction to produce a dislocation; flexion alone or adduction alone may cause a subluxation, but never a complete dislocation. In chronic infections of the hip, complete dislocations do not occur, because the muscle spasm is not sufficiently severe. The condition can be prevented in both paralytic and arthritic patients by preventing adduction and flexion. Corrective measures, on the whole, are unsatisfactory, because the dislocation in so many cases is associated with other irreparable damage.

[ED. NOTE.—We are inclined to be more optimistic than Jones appears to be in respect to the value of corrective operations in spontaneous dislocations of the hip. In the paralytic patients the shelf operation, with suitable modification, affords a means of stabilization. In the tuberculous cases one must employ an ankylosing operation to obtain the same end. Dislocation resulting from septic infection constitutes the most difficult problem on account of the risk of a flare-up of an old septic process, but even here by means of carefully planned and executed operations it is often possible to obtain an ankylosis.]

(To be Continued)

CORRECTION

In the article by Dr. Deryl Hart, entitled, "Intracystic Papillomatous Tumors of the Breast, Benign and Malignant: Analysis of One Hundred and Twenty-Four Cases (ARCH. SURG. **14**:793 [April] 1927), some of the legends, beginning with figure 6, are incorrectly placed. The legend under figure 6 should be under figure 18, and each of those following should appear under the preceding illustration, i. e.: the legend under figure 7 should be under figure 6; that under figure 8 should be under figure 7, and so on.